Pediatrics Supplement

Contributed by

The American College of Osteopathic Pediatricians

Volume 8
Number 1

December, 1955
Pages 1 to 16

PEDIATRICS SUPPLEMENT

The American College of Osteopathic Pediatricians

Editorial Office, 212 East Ohio St., Chicago 11.
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The Common Cold and Sinusitis*

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The Commission on Acute Respiratory Diseases,[1] in studying the etiology of the common cold, has experimentally transmitted minor respiratory illnesses to human volunteers and has demonstrated that at least two filtrable agents, presumably viruses, can induce minor illnesses in man. These illnesses are distinct disease pictures. The first has a short incubation period of 1 to 2 days and is characterized by sneezing, nasal obstruction, coryza, and cough. The second has a 5- to 6-day incubation period and is characterized by sore throat but few nasal symptoms. Following this study by the Commission, it was felt that proof is now available that at least two filtrable agents or viruses may induce a minor respiratory illness. If this is true, it can be stated that the common cold is a communicable disease. The number of colds per person in the United States is approximately three per year.[2] This includes those persons who have immunity lasting for only a few months as well as those who apparently have a much longer immunity or practically a complete immunity, those who never seem to get a cold.

Now, what is the common cold? After the Region I meeting of the American College of Osteopathic Pediatricians in Boston a year ago, in which a symposium was held similar to the one today, we were satisfied that we had proof that the common cold is not rhinitis, pharyngitis, otitis, sinusitis, tracheitis, or bronchitis, but that it is an inflammatory reaction of all the mucous membranes of the upper respiratory tract due to a specific virus or viruses which, at present, will have to be known as the common cold virus.

Upon invasion of the upper respiratory tract there is first edema of the submucosa, followed by infiltration with white blood cells. The surface cells slough off and appear necrotic. This process may be severe and may leave but a single layer of cells in the basement layer of the mucous membrane. The cells then proliferate and again resume their columnar shape in the final stages of repair. These findings have been confirmed by various workers, and the reaction has been found to be quite pure in small infants in whom the process has been definitive and acute. Considering that the entire upper respiratory tract is lined with a contiguous mucous membrane, I think that it is practically impossible to believe that the common cold virus upon invasion of this mucous membrane will not or cannot spread throughout its entirety and into the sinuses, ears, and trachea.

There are exceptions to this, of course. In very early infancy the common cold is usually mild and characterized mostly by nasal symptoms of sneezing and some congestion of the nasal mucous membrane. The reason for this is that the infant still possesses immunity from the mother, the sinuses are not yet open, and there is good eustachian tube drainage. There is usually very little or no fever, and in 5 or 6 days, the child is perfectly well again. This is about the mildest form of the common cold seen in infancy and it is usual only in the first month or two of life.

In infants between 2 and 6 months, the common cold usually manifests itself with a slightly greater extension along the mucous membrane of the upper respiratory tract, and then there is a mild degree of swelling in the throat without much redness, but edema is apparent. The pharynx is edematous, the child may seem a little hoarse, and he voluntarily reduces his food intake for a day or 2. The cold lasts a little longer than a week, but there is usually no complication.

Between the ages of 6 months and 2 to 3 years the common cold becomes more severe in type. There is a greater degree of fever and more cough and anorexia. The inflammatory reaction is more extensive, going through the eustachian tubes to cause otitis media, extending into the sinuses that are now opening to cause sinusitis, extending down the pharynx into the trachea to cause tracheitis and laryngitis and down a little farther to cause bronchitis. Pneumonia as a complication of the common cold is at its peak in children 2 years of age. The extension of the viral inflammation along the mucous membranes reduces the membranes' ability to combat infection, and secondary invaders enter. The longer this situation persists, the greater are the chances that otitis media, sinusitis, and pneumonia will complicate the cold, and the graver is the patient's condition.

In the early infant years, parents as well as doctors may become distraught with the problem of the succession of these colds. Of course, the child must build up his own resistance with the help of the physician and the parents. As his resistance or antibody response becomes greater and the child becomes older, his colds become more adult in pattern and, although they assume the afebrile state of the young infant, they also assume the headache, anorexia, malaise, and aching which are more common in the adult type.

The older child begins to complain of throat symptoms, a complaint which is very rare in children under 4 years of age, possibly because they are unaware of it. They have dysphagia, but do not perhaps associate it with pain as such. After the age of 4 years more common complications of the common cold such as sinusitis begin to occur. Abdominal symptoms may be present, although these also may be noted even in the very young infant. The child may have diarrhea or may be vomiting, and upon inquiry the physician may discover that other members of the family have colds; the gastrointestinal upset in this case may be the first symptom in the child. The first symptoms of the common cold in the newborn or in the first 6 months may be only those of a gastrointestinal upset; in the older child, anorexia, abdominal pain, and vomiting are quite common.

This syndrome can also be appendicitis. We have frequently noted in our hospital that appendicitis sometimes seems to run in epidemics along with colds. Brennemann[3] has also noted this; in his experience, more than half of all cases of appendicitis apparently accompanied or followed upper respiratory infection. It has also been reported that appendicitis occurred eight times more often among 16,000 students at the University of Wisconsin during epidemic periods of upper respiratory infections than during other periods.

*Presented at the National Osteopathic Child's Health Conference and Clinic, Kansas City, Mo., April 4, 1955.

This syndrome of vomiting and abdominal pain with or without diarrhea has often been called "intestinal flu." Diarrhea associated with respiratory diseases is common in the infant wards of many hospitals, and I think it is extremely important to remember that diarrhea and vomiting in the newborn are rarely due to formula difficulty. Therefore, the physician should look first for some communicable infection of the upper respiratory tract.

Sometimes a mother states that her infant or child has had a cold ever since he was born, that he came home from the hospital with a cold and still has it, or that he no sooner gets over one cold when he catches another. His nose is always running, he cannot breathe comfortably, and he cannot eat or sleep well. Such a condition is usually not a cold; it is allergic rhinitis. Allergic rhinitis may not remain pure rhinitis for long and may develop into allergic sinusitis or allergic otitis.

In infants with allergic rhinitis the membrane in the nose is pale, swollen, and watery looking. The discharge is shiny and stringy, and upon staining it is found to contain many eosinophils. If the discharge persists in spite of the use of nose drops, the case should be investigated as soon as possible in an attempt to determine the allergen that is responsible.

The common cold must be differentiated from influenza, and this should not be difficult. The influenza patient is toxic, flushed, and feverish; the pharynx is congested, and there may be epistaxis. The onset of influenza is sudden. Acute infectious and contagious diseases such as measles and pertussis must also be differentiated, and poliomyelitis should be considered.

I shall discuss treatment according to age groups. In the newborn period where nasal congestion is the symptom, I very often use only osteopathic manipulative treatment. Occasionally I administer some Privine nose drops and assure the mother that the child will be all right in from 3 to 5 days, and tell her to elevate the baby's head and watch his temperature carefully so any complication can be anticipated.

Tracheitis or tracheobronchitis is the most common complication in infants between about 2 and 6 months. In addition to osteopathic manipulative therapy, I treat the nasal symptoms and recommend elevating the head of the bed and increasing the room humidity with a vaporizer, and prescribe aspirin in doses of 1 grain per year of age every 4 hours or as necessary to make the child more comfortable. I watch constantly for the complication of otitis media which begins at about 6 months of age and also for pneumonia which may accompany it. These conditions of course are treated with antibiotics as indicated.

A word should be said for the various medical treatments that have been tried or are being used in many areas at the present time for the treatment of the common cold. Gamma globulin has been mentioned as a possible source of passive immunity not only to measles and poliomyelitis but also to the common cold. Studies indicate that there is some value in the use of gamma globulin in colds.[4]

Sinus involvement is the greatest problem in children between 6 months and 2 to 3 years of age. A chronic infection may be confined to the sinuses or there may also be concurrent bronchitis. Whether bronchitis results from actual drainage from the sinuses down to the bronchial tract or as a contiguous spread along the membrane is still unknown, but the occurrence of a sinobronchitic syndrome cannot be denied. In New England there is a tendency to treat sinobronchitis as sinusitis complicating the common cold, with the idea that if the sinusitis as such is treated the bronchitis will take care of itself.

It is necessary to understand the developmental anatomy of the accessory sinuses in order to be able to appreciate why the common cold in infancy and childhood is a panmucous membrane infectious disease of the upper respiratory tract. The sinuses, after they are developed from the facial bones and filled with air, are lined with mucous membrane that is directly continuous with the nasal mucosa. The sinuses first become differentiated as evaginations of the nasal fossae during the fourth fetal month. In childhood, the ethmoid cells are so closely related to the frontal sinuses that I will consider their pathology together. The sphenoid sinuses are difficult to visualize, but they are involved just as much as the maxillary and/or the frontal sinuses, since the membrane is continuous from the nasal cavity in through the sinus pouchings. These protrusions of the nasal fossa during the fourth fetal month then become actually differentiated by the local evagination of the nasal mucous membrane, with concurrent reabsorption of the overlying bone. The continued growth of these pouches and the concomitant regional destruction of bone result in the formation of paired pneumatic cavities lined with evaginated nasal epithelium. It is most important to know how early in life these paranasal sinuses may become involved. Of course, they cannot become involved in a clinical entity with infection until they are pneumatized and developed.

One of the best ways to appreciate the development of the nasal sinuses, particularly the frontals and the maxillaries, is to observe it on x-ray film, from birth through to 12 years of age. A striking difference will be noted in the size and shape of these cavities, and variations will be seen in different age groups and in different individuals and on the two sides of the same individual. The roentgenographic interpretation of the findings in the paranasal sinuses is much less accurate in infants and children than in adults, because in children the cavities are smaller and the margins of healthy sinuses are often indistinct. There is a lesser contrast in density in the cranial bones themselves so that the sharp delineation needed in a good x-ray is absent. If these x-rays of normal sinuses are then compared with a series with pathologic findings, a better idea can be obtained of what to expect of sinusitis in the infant and child.

Necrotizing ethmoiditis, with great swelling of the orbital tissues which are red, angry, and often discharging, must be differentiated from an infected insect bite or suppuration of the lacrimal duct. Necrotizing ethmoiditis is not so common since the advent of antibiotics. It occasionally does occur and must be treated promptly with sufficient antibiotics and drainage. However, in the vast majority of cases of sinusitis in infants and children, it is the highly developed maxillary sinus that is chiefly involved. As I previously mentioned, there may also be hypersensitivity of the mucosa owing to a familial predisposition to chronic rhinitis in which allergy is the etiologic factor. There is definitely an allergic factor in silent sinusitis, but in the vast majority of cases sinusitis is caused by a common cold.

The viruses of the common cold probably always infect the sinuses, and then curing of the sinusitis or healing of the inflamed sinus mucous membrane lining is delayed by a superinfection. This delay, of course, is also favored by nasal obstruction and insufficient

ventilation of the sinuses. Adenoid vegetations interfere with nasal breathing and normal nasal drainage. The probability cannot be ruled out that maxillary sinusitis occurs first, before the lymphoid swelling, and secondarily, as a result of the drainage of secretions during the night. This produces a swelling of the lymphoid tissue at the pharyngeal ring with secondary infection.

Children are not usually disturbed in their normal activities by the minor discomforts of sinusitis, but some mouth breathing at night, increased nasal secretion, and an irritative night cough will be noticed. These symptoms are generally attributed to tonsil or adenoid hypertrophy by the layman and, until a short time ago, by many general practitioners. After correction of these difficulties the susceptibility to colds and the hacking cough would continue. Further investigation, particularly of the paranasal sinuses, and x-rays of the lungs would reveal what I prefer to call the sinobronchitic syndrome, which probably was the original etiologic factor, with the adenoid or lymphoid involvement secondary.

Interpretation of these roentgenograms requires considerable experience, and when the maxillary sinuses in particular are found to be cloudy, an attempt should be made to shrink the nasal mucous membrane in order to permit drainage, instead of immediately performing an exploratory surgical irrigation. Such a procedure should be performed only by a specialist, because there is the considerable danger of damaging the orbit and even the intracranial space in infants and children.

The most reliable diagnostic method for silent sinusitis is roentgenography, and the roentgenologist should routinely check the maxillary sinuses when x-raying the chest, which is the procedure used in the clinic at the Massachusetts Osteopathic Hospital. The high incidence of positive findings will be astounding, and with some experience, the cloudiness of the maxillary sinuses will be recognized early, treatment instituted at a reasonably early date, and chronic sinusitis with the sinobronchitic syndrome can be cured.

Therapy for these cases is divided into surgical and nonsurgical. A nasal mucous membrane–shrinking solution, such as Privine or Neosynephrine, may be tried, using nasal packs, and osteopathic manipulative therapy should be applied to the cervical-dorsal area, and an attempt should be made to provide better lymphatic drainage through soft tissue manipulation. The purpose of treatment, of course, is to produce drainage. Chemotherapy or antibiotic therapy is also utilized. Negative pressure irrigations of the nasal cavity are indicated in order to try to produce a sucking effect from the maxillary and frontal sinuses. A change of climate may be advocated which may provide great relief to the child. Surgery is performed only as a last resort, the purpose again being to provide open drainage into the sinus areas by surgical irrigations.

In conclusion, the common cold is the most common infectious and contagious disease in childhood, and it is accompanied by an extremely annoying and symptom-producing complication, pansinusitis, accompanied by the sinobronchitic syndrome. It is particularly important that the general practitioner be ever watchful for possible infections of the paranasal sinuses following or accompanying the common cold in children.

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Diagnosis and Treatment of Sydenham's Chorea, With Presentation of a Case

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Differentiating Sydenham's chorea from similar or related conditions may become an important consideration in diagnosis. Several interesting features were proposed by the case presented which emphasized the controversial aspects of this disease and underlined the fact that Sydenham's chorea still is a problem confronting the medical profession.

The patient admitted to the hospital was a 10-year-old white male, hyposthenic, and mentally alert. The diagnostic impressions on admission were chorea, conversion hysteria, and focal encephalitis. He was admitted for study because of increasingly severe and involuntary body movements of 5 years' duration. The symptoms began when he started school at 6 years of age and were more severe in winter than in summer. At first, there was merely biting of the lips. In time, progressive spreading of spasmodic movements occurred down the body and extremities, finally involving the diaphragm and resulting in an involuntary barking noise. When the child was able to relax, the twitching seemed to abate momentarily; it disappeared when he was sound asleep.

The child was sent to a child guidance clinic for treatment, but there was very little, if any, improvement in his condition. An electroencephalogram had been made a year prior to admission to the hospital, but it was noninformative.

His childhood diseases had consisted of measles, mumps, chickenpox, and pertussis. There was no history of injury at birth. The boy had developed normally until the onset of symptoms. His brother, aged 17, had no history of any disorders. The rest of the family history was negative except for the presence of diabetes mellitus in one grandparent. The patient averaged two to three colds a year with occasional nose bleeds. His appetite had been poor but there had been negligible weight loss. His height on admission was 4 feet, 4 inches, and his weight was 64 pounds.

On physical examination, it was found that the tonsils were present, but they were not remarkably enlarged or diseased. There was minimal cervical adenopathy and no cardiac murmurs or cardiac enlargement. The rest of the physical examination was noncontributory except for the neurologic findings.

Neurologic examination by a neuropsychiatrist revealed an alert and cheerful child with irregular speech consisting of slurring, stammering, blocking, and explosive outbursts, apparently due to motor involvement. The thought content, however, was normal. The motor activity was grossly abnormal, characterized by diffuse, bizarre, spasmodic, and athetoid movements of the entire body, including the diaphragm. The movements resulted in grimacing, gesticulating, and nictitating activities which were not stereotyped or ritualistic in nature or succession.

Under Amytal sodium sedation, athetosis diminished in intensity but it did not diminish in rate until light anesthesia was attained. No alterations in emotions, attitude, or behavior resulted during this period.

There were no other pathologic findings of the nervous system.

The following are the results of laboratory tests:

Erythrocytes 4,750,000

Hemoglobin 103.5 per cent

Leukocytes 14,580: 69 per cent polymorphonuclear cells, including 3 per cent stab cells; 37 per cent lymphocytes; and 1 per cent eosinophils

Blood serologic test—negative

Sedimentation rate (on 3 occasions) 5 mm. per hour

Spinal fluid examination—essentially normal

Urinalysis—essentially normal.

Chest and cranial x-rays were negative, and electrocardiographic findings were essentially normal.

The symptoms, history, and laboratory findings did not indicate the presence of an active or chronic infection. There were no focal symptoms of localized brain lesions such as brain abscess, tumor, or trauma. The patient's symptoms did not suggest epilepsy, especially in the absence of a history of trauma or familial history of epilepsy. Psychiatric evaluation ruled out major psychiatric conditions. The patient's history and actions resembled very greatly those in Sydenham's chorea. Treatment was directed toward that condition.

Treatment on admission consisted of administration of ACTH, Dilantin sodium, and mild sedatives when necessary. No appreciable improvement was noticed. The physical examination, history, and laboratory findings did not substantiate a diagnosis of rheumatic fever.

After neurologic consultation and evaluation of the laboratory findings, the treatment was changed. Reserpine and chlorpromazine therapy was then instituted in combination with Dilantin sodium. This combination of drugs resulted in progressive improvement of the patient's condition. The child was placed in a room by himself. Later, even though rheumatic fever was proved not to exist, Achromycin and a sulfa drug were added with high dosages of vitamins. Oranixon was also administered with the idea of combating muscle spasm. At night, strong doses of barbiturates were given in order to induce sleep and, as the patient's condition improved, the dose was reduced. When chorea diminished, osteopathic manipulative treatment was begun.

After 1 week of therapy the patient improved so remarkably that he was discharged from the hospital.

At home treatment continued, as outlined, for another month when the therapy was limited to administration of reserpine and chlorpromazine. His speech improved so that there was only an occasional slur, and the convulsive movements had diminished until there were only isolated body twitching and diaphragmatic spasms. When the patient's condition warranted, educational instruction was begun. Interestingly, the patient revealed an unusual liking and aptitude for a musical instrument, and playing it was encouraged. The child, after several more months of improvement, was allowed to return to school and begin a slow adjustment to his environment.

DISCUSSION

Sydenham's chorea still remains a diagnostic puzzle. Usually it is classified with the rheumatic diseases, in spite of the fact that there are reports of cases due to syphilis, brain lesions, inherent predispositions, and psychogenic factors, and that there are congenital types.[1] Many studies have been made in the British Isles aimed toward investigating factors which might be expected to lead to such disturbances and attempting to correlate the mental and emotional attributes and make-up of patients. The conclusions, based on studies of environmental backgrounds of patients, show that if there are sufficiently severe or long continuing psychologic disturbances from which a child can find no satisfactory escape, hyperkinesis or hypotonia characteristic of chorea can be produced through corticothalamic exhaustion.[2] The investigators noticed that the mothers of children with chorea usually were overconscientious and often had strong scholastic or social ambitions for their children. Furthermore, they maintain that the nature of the association between chorea and rheumatism is, in all probability, a common etiologic factor. The two conditions need not occur at the same time and, in all probability, chorea usually leads to rheumatism rather than the reverse.

Another clinic group[3] showed in a series of patients that not all chorea cases were associated with rheumatic fever and that, specifically, an initial episode of chorea in a person with a normal sedimentation rate and no other evidence of rheumatic fever is probably not due to rheumatic fever. These cases, according to this clinic, should be referred to as chorea (Sydenhams), cause undetermined. A diagnosis of chorea associated with rheumatic fever should be made with certainty only when there is other evidence of rheumatic fever and recurrences.

Another group of investigators[4] attempted to classify chorea etiologically as rheumatic chorea and nonrheumatic chorea. The distinction between the two is based on the fact that the sedimentation rate is more often elevated in the rheumatic episodes, and psychic disturbances are more commonly associated with the nonrheumatic group. They modify this conclusion by saying:

> One is still not wholly justified in assigning to psychogenic disturbances a major etiological role in the causation of Sydenham's chorea, but we feel that this factor is worthy of further consideration and study, especially in non-rheumatic chorea. . . . Erythrocyte sedimentation rate determinations as well as psychogenic factors may be of aid in the differentiation of rheumatic and non-rheumatic chorea, but their value is limited.

According to their results, of the cases with elevated sedimentation rates, 65.9 per cent had rheumatic complications while only 30 per cent of the cases with normal sedimentation rates had rheumatic complications.

However, these results merely show a tendency rather than a hard and fast rule.

An eminent neurologist[5] classifies chorea as an extrapyramidal tract disease occurring most frequently between the ages of 5 and 16 years, more often in females, and appearing usually in the spring. There is commonly a familial history of neuropathic tendencies, alcoholism, epilepsy, various tics, and insanity. A rheumatic history is found in 72 per cent of cases of chorea, but 86 per cent of rheumatic fever cases have no history of chorea.

It has been noted that fright, excitement, and worry usually precede the onset of chorea, and fear and excitement may precipitate an attack. Likewise, infections such as scarlet fever and diphtheria can precede an attack.[5]

The onset is usually abrupt, although there may be a history of previous minor movements or of a recent or repeated sore throat. Symptoms may begin with grimacing and then become progressively worse with more profuse movements of brief, discrete, abrupt, unsustained, irregular, and nonstereotyped forms. Quick movements of the head, shoulders, and arms and tossing of the head are commonly found. Voluntary movements are of necessity performed abruptly and suddenly. Grunting noises and indistinct and incoordinate speech are characteristic. Symptoms disappear during sleep. Muscular weakness can occur even to the point of hemiplegia.

Chorea patients are irritable, poor sleepers, and emotionally unstable. They are regarded as problems in school. Many are highly intellectual and highly imaginative, with superior school performances, but they daydream and are introverted. Recurrences of attacks are common over a number of months or years.[5]

Laboratory tests are not particularly helpful in diagnosing chorea, since in the rheumatic type the laboratory findings will be similar to those of rheumatic fever, and in the nonrheumatic form the results will not be positive as compared to the rheumatic type.

Just as the etiology of the disease is puzzling, so is the treatment. It is maintained that chorea is a self-limiting disease with no specific therapy. Treatment is therefore aimed toward eliminating any focus of infection, improvement of the nutritional status of the patient, psychologic readjustment, and controlling the severity of muscular motions and protecting the child against injury.[6]

One of the old-time medications used in the treatment of chorea is arsenic in the form of Boudin's solution or Fowler's solution. This type of treatment has been proved to be of no value, but it is still in use. Nirvanol, one of the hydantoin drugs, has been used for years.[7] This drug is credited with shortening the duration of the chorea attacks. However, the toxicity of Nirvanol is so great that its use is not feasible. Many other drugs have been tried with little or no success, such as antipyrine (which is very toxic), sodium salicylate, aminopyrine, Chloretone, sulfanilamide, intravenous magnesium sulfate, thiamine hydrochloride, and pyridoxine. Pyridoxine showed great promise in one clinical study[1] since it can be given in large doses without any side-reactions; however, to my knowledge, no further studies have been made.

Other drugs which were used in the past include epinephrine, thyroid preparations, parathyroid preparations, electrolyzed colloidal silver, iodine, gentian violet, methenamine, and 1 per cent phenol. More recently, the ketogenic diet was used in the treatment of chorea, but this too has fallen into disuse.

A popular treatment has been fever therapy, using insulin, sterile milk, or typhoid as the pyrexic agent. The duration and the recurrence rate of chorea have been decreased with this therapy, but the danger and the complexity of fever therapy seem to be drawbacks to its use.

Actually, the most important aspects in the treatment of chorea are bed rest, quiet, sympathetic environment, and parental understanding. During the acute phase, mild sedatives in the daytime and heavier nightly sedatives to induce sleep are very important. Phenobarbital, pentobarbital, and paraldehyde are good for this purpose.

If chorea is associated with rheumatic fever, therapy is directed toward the treatment of the latter, including administration of penicillin and vitamins, bed rest, and so on. In the nonrheumatic type, psychotherapy is indicated. Hot packs and warm baths combined with the ketogenic diet are still used, but even though this treatment may be helpful, it is not always easy to apply. Recently, with the discovery of various steroids, ACTH and cortisone have been tried in the treatment of chorea. One clinic[8] has found no value in their use, and yet another[6] concluded that they show promise. However, steroids at no time have given any spectacular results. They apparently give better results in the rheumatic type of chorea.

When Benadryl was introduced, it in turn was experimented with, but no beneficial results were obtained.

In the case described, a new group of drugs was used, mainly chlorpromazine, Ornixon, and Dilantin sodium, although the last is well known as an antiepileptic drug. Fairly good results were obtained but no definite conclusions can be drawn from this isolated case. However, this may give some incentive to explore these drugs further. It is quite possible that a new field of experimentation can be started.

The prognosis of chorea varies with each individual case. As was stated previously, Sydenham's chorea is a self-limiting disease. With proper care, patients improve, but if the etiologic factors persist, a recurrence can develop. If there have been pre-existing diseases such as encephalitis or schizophrenia, the follow-up social adjustment of these children after recovery from chorea will be poor.[9] Social and emotional deprivation, a low IQ, profound reading disability, prolonged serious illness that interferes with normal education and personality development, and a possible permanent damage or long-continued organic brain disturbance by the rheumatic process are reasons for a guarded prognosis. Many children who have recovered from chorea have shown a progressive falling off of their IQ's over a period of a year. Social adjustment in the main depends on the quality of pre-existing environmental background and the existence of behavior problems prior to the onset of chorea.[6]

SUMMARY AND CONCLUSIONS

A case of Sydenham's chorea has been presented in which it was proved that the patient had organic brain disease but no clear-cut history of an antecedent infection and a more or less chronic recurrence of the condition over several years. Several innovations in treatment were attempted, with a fairly successful outcome which should be an incentive for further research.

A comprehensive perusal of the literature has shown that the etiology and treatment of Sydenham's

chorea are not clearly understood. Statistics reveal a general decline in the incidence of the disease during the past 2 decades[1] which is believed to be due to improved standards of living. This implies that good general nutrition might play an important part in the prophylaxis of this disease. Since there is definite evidence that the etiologic background of rheumatic fever and Sydenham's chorea is similar and one condition may precede the other, the two are frequently considered together.

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Amenorrhea of the Adolescent*

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PHYSIOLOGY OF MENSTRUATION

In order to understand better the problem of amenorrhea, it is necessary to review the physiologic basis of menstruation and the occurrence of the menarche in the normal adolescent.

The hypothalamus is the seat of centers which integrate the activity of the central and autonomic nervous systems and the endocrine glands. It initiates activity in the anterior pituitary gland which in turn secretes the gonad-stimulating hormones: (1) follicle stimulating hormone (FSH), (2) luteinizing hormone (LH), and (3) luteotrophic hormone (LTH).

These gonadotrophins activate the ovaries to produce estrogen and progesterone. These steroid substances stimulate the growth of the endometrium. When the ovarian hormones reach sufficiently high blood serum levels the production of pituitary gonadotrophins is suppressed temporarily. This results in prompt fall of estrogen and progesterone production.

Consequently the endometrium, suddenly deprived of its hormonal support, breaks down and bleeding results—the phenomenon of menstruation.[1]

The average age for the occurrence of the menarche is 13 years. Its occurrence at any time between 9 and 17 years is considered within normal limits. The appearance of the secondary sex characteristics, enlargement of breasts and genitalia and appearance of pubic and axillary hair, indicates maturation, and development begins about a year before the first menstruation. There is also an increase in somatic growth—lungs, liver, kidneys, and skeleton.

CLASSIFICATION OF AMENORRHEA

Amenorrhea should be looked upon as a symptom. Amenorrhea is the absence or abnormal stoppage of the menses. Absence of menstruation for 3 months or more is generally considered to be amenorrhea. Amenorrhea can be further classified into primary, in which menstruation has never occurred, and secondary, in which the menses have appeared but have been suppressed. During pregnancy, when cessation of menstruation is a physiologic phenomenon, amenorrhea is not considered abnormal. Differentiation between pregnancy and secondary amenorrhea must always be made.

The majority of cases of secondary amenorrhea have an endocrine basis. Fluhmann[2] classifies secondary amenorrhea into five general groups: physiologic amenorrhea, cryptomenorrhea, amenorrhea of uterine origin, postcastration amenorrhea, and functional amenorrhea.

DIAGNOSIS

The taking of a thorough history will be helpful in diagnosing the type of amenorrhea. The menstrual history of other members of the family should be noted including the occurrence of sterility and the character of the periods from the menarche through to the menopause. Any emotional disturbance, past or present, in the life of the patient may have a bearing on glandular functioning. Severe illness with hyperpyrexia may damage the hypothalamic area. Operations such as that for a ruptured appendix may possibly cause pelvic adhesions.

A complete physical examination including laboratory studies is a must. A great teacher once said that more mistakes in diagnosis are made by not looking than by not knowing. When the possible causes of secondary amenorrhea are considered, the importance of minute searching by every possible method can be realized.

The general appearance of the individual, especially the distribution of adipose tissue, may give an immediate clue. The secondary sex characteristics will reveal a tendency toward masculine habitus. Body build, breasts, distribution of hair—particularly the pubic hair—size of the clitoris, size and configuration of the vagina and of the uterus, so far as can be ascertained, are particularly pertinent in this regard.

*Presented at the Twenty-Third Annual National Osteopathic Child's Health Conference and Clinic, Kansas City, Mo., April 6, 1955.

The examination should be conducted in such a way as to avoid possible psychic trauma. One of the reasons the patient has come to the physician in the first place is because she is different from others, which makes her unhappy. She will understand the necessity for a thorough examination when it is explained to her that such an examination is needed to learn why she is different from other girls. The importance of a thorough examination may have to be stressed when rupture of the hymen is necessitated by performance of certain laboratory procedures. In some cases, the patient does not receive the best care if these investigations are omitted. However, in most instances, there is justification in the use of simple procedures first and reservation of the more complicated ones such as biopsy until after others have been performed. Of course, the physical findings will dictate to a certain extent what diagnostic laboratory technics will be used first.

The following laboratory procedures are indicated: The usual complete blood count and urinalysis are basic and can give the answer immediately in some cases. A basal metabolic rate study (not just one test), x-ray of the sella turcica, a 5-hour glucose tolerance test, vaginal smears, and, in some cases, endometrial biopsies will follow, in descending order of frequency.

Cytologic studies of the vagina will reveal the presence or absence of adequate estrogen stimulation. A freshly prepared slide of the vaginal secretions is stained with 1 per cent pinacyanole for 30 to 60 seconds, or other standard procedures, such as the Papanicolaou, are used. A majority of cornified or precornified epithelial cells with dense pyknotic nuclei is indicative of estrogen activity. Conversely, small round cells containing large nuclei indicate estrogen deficiency.

Endometrial biopsies, particularly if repeated weekly, will reveal the status of ovarian activity. If the endometrial biopsy shows hyperplasia it indicates an imbalance in the pituitary-ovarian relationship. Gonadotrophins should not be prescribed for these cases.

The urinary gonadotrophin titer will aid in differentiating primary ovarian failure from ovarian failure secondary to hypofunction of the anterior pituitary. When the level is low the patient will respond to cyclic courses of combined anterior pituitary gonadotrophic hormone and chorionic gonadotrophin after priming with estrogens. The normal gonadotrophin titer will be 6 to 50 mouse units. Less than 6 indicates pituitary failure and more than 80 indicates ovarian failure.[1] Estrogens are more effective in amenorrhea due to ovarian failure.

Evaluation of the thyroid function can be made easily by studying basal metabolic readings (a random basal metabolism test is not too reliable) and blood cholesterol levels. Elevation of the blood cholesterol indicates hypofunction of the thyroid. Administration of a small amount of dessicated thyroid daily is effective in many cases of amenorrhea; usually ½ to 1 grain is used initially and may be increased if the patient's pulse rate and weight are carefully watched. Two grains daily are generally the maximum unless the basal metabolic rate was extremely low. It is important not to increase the dosage earlier than 2 to 3 weeks after administration is begun as this medication is cumulative, more so in some patients than in others.

The occurrence or absence of ovulation can be determined with reasonable certainty by the basal temperature chart. The body temperature, recorded daily during the menstrual cycle and plotted, shows an abrupt dip, usually in the midmenstrual period (thirteenth to seventeenth day). The curve rises sharply again and reaches a level about 0.8 above normal; this level is maintained until the menstrual flow begins, at which time the temperature returns to normal. The girl whose temperature is constant is probably not ovulating.

Ovulation is also followed by the appearance of pregnanediol, a derivative of progesterone, in the urine. Its detection is therefore taken as evidence of ovulation and fixes approximately the time of its occurrence.

In conjunction with these diagnostic procedures I shall now discuss in more detail the five types of secondary amenorrhea.

SECONDARY AMENORRHEA

Physiologic amenorrhea is the absence of menstruation as the result of a physiologic disturbance. It might be thought of in terms of conservation of blood, such as absence of menstruation in a girl who has dieted strenuously and whose metabolism is out of balance from a nutritional standpoint. The converse of this also serves as an example, for instance, the obese patient who diets sensibly and loses weight slowly enough that her metabolism is not upset. In this case, the strain of the extra weight is eliminated, and in many cases the menarche or resumption of normal menstruation results.

Cryptomenorrhea is prevention of the menstrual flow as a result of a mechanical obstruction in the vaginal or cervical canal. Most cases occur during puberty and are congenital in origin, for example, as the result of an imperforate hymen. Cases occurring later in adult life result from (1) postpartum masses, (2) trauma, (3) stricture formation following cervical or vaginal inflammation, (4) plastic surgery, (5) injury following radium therapy or cauterization, and (6) neoplasm. Since the ovaries and endometrium are functioning cyclically, this condition is not true amenorrhea.

Amenorrhea of uterine origin results when the uterus has been removed or injured but the ovaries are intact.

Postcastration amenorrhea occurs when the ovaries have been removed or have atrophied; in younger women, premature menopause results.

Functional amenorrhea is suppression of the menses in women whose uterus and one or both ovaries are present. Systemic disease such as tuberculosis or decompensated heart disease, tumor of the hypophysis, or endocrine disturbance may be the cause. In the majority of cases of functional amenorrhea an endocrine disturbance is probably at fault.

As was stated earlier, the anterior pituitary, or anterior hypophysis, as it is sometimes called, exerts the "kick-off" influence in menstrual activity. The anterior pituitary is in turn sparked by the hypothalamus.

What are some of the things that can interfere with the function of the pituitary? I want to list first the osteopathic structural approach. Dr. Reed[3] brought out so well the relationship between structure and function, and I know of no place where it is more definitely illustrated than in this inter-relationship between the nervous system and the endocrine system. This applies to each of the glands, and it has long been accepted that a disorder of one will automatically affect the whole system. It is a foregone conclusion that vitality is not good in a girl whose structure is not good.

Other factors in deficiency of the hypophysis are trauma, hemorrhage, infections, and tumors, and Fluhmann[2] also considers an inherent constitutional defect as another possibility. Frölich's syndrome, with its obesity, hypertrichosis, and genital dystrophy, and Simmond's disease, manifested by emaciation, cachexia, asthenia, and amenorrhea, may result.

Either hypofunction or hyperfunction of the thyroid may disturb the menstrual cycle. Fluhmann[2] believes hyperthyroidism is more common in amenorrhea, and, experimentally, hyperthyroidism inhibits gonadotrophic and estrogenic hormones, but that "a considerable number of amenorrheic patients . . . may show a lowered basal metabolic rate, but in this case perhaps both findings depend on a common factor and one is not necessarily the result of the other." X-ray estimate of bone age gives much information on thyroid function. The pisiform is the last of the carpal bones to ossify, and this usually takes place shortly before the beginning of the menarche.

Hyperplasia of the adrenal cortex and certain cortical tumors produce an endocrine disturbance resulting in hypertrichosis with a masculine distribution of hair, enlargement of the clitoris, and atrophy of the uterus with amenorrhea. In one series of 123 patients with Addison's disease eleven had amenorrhea.[2]

The pancreas and its secretion of insulin may get out of balance. It is known that insulin depresses ovarian function.

The ovaries are of course the focal point in menstrual disturbances. Removal of a unilateral arrhenoblastoma, for instance, usually restores menstruation when amenorrhea has been present. Whether or not the ovaries are present, bleeding can be induced by adequate estrogen and progesterone stimulation when the uterus and endometrium are intact.

TREATMENT

When cytologic studies of the endometrium and vagina indicate low intrinsic estrogen levels, progestin therapy alone is insufficient to produce bleeding. Instead, treatment is first directed to the endometrium by administering estron sulfate, 1.25 mg., or its equivalent, daily for 20 days, followed by progesterone, 10 mg. intramuscularly each day for 3 to 5 days, or orally, 30 mg. three times a day for 5 days, or as anhydrohydroxyprogesterone, 10 mg. three times daily for 5 days.

Simultaneous oral administration of estrogen and progesterone has proved slightly superior to progesterone alone, but does not take the place of priming when intrinsic estrogen levels are inadequate.

If the uterus is infantile, estrogens will often help. Induction of cyclic bleeding is worth while if amenorrhea is causing extreme emotional anxiety. Treatment is also indicated for women who desire children as the chance for conception is improved.

Frequently there is a report of pregnancy during amenorrhea. Here is proof that ovulation and the subsequent development of a corpus luteum may take place even after long periods of amenorrhea. In such a case fertilization of the ovum prevents the flow which was about to take place.

Stimulating doses of deep x-ray therapy produce good results in some patients, but since the margin between the safe stimulating dose and the dangerous one is narrow, this treatment should be administered only by the specialist.

Menstruation is not a type of purgation to rid the body of detrimental substances, and there is no accumulation in the body of wastes when the menses do not occur. Relieving the mind of the patient of this superstition and ridding her of apprehension are often beneficial. Sometimes this psychic relief alone will affect the nervous system and, therefore, the whole mechanism sufficiently to relax the patient and result in normal menstrual function.

There is no question in my mind that the results of osteopathic manipulative treatment, if tabulated systematically, would prove superior to those of other methods. This is the confidence resulting from 20 years of performing osteopathic manipulation.

In the embryo the ovaries develop near the kidneys in the upper abdomen and, as they descend into the pelvis, drag their blood and nerve supply with them. Therefore, visceral innervation is from the lower thoracic and upper lumbar segments, a transitional area of the spine where the human is especially prone to strain and osteopathic lesions. The superior facets of the twelfth thoracic vertebra are usually thoracic in type, while the lower facets are of the lumbar type.

Normalization of all spinal lesions will bring amazing results. No two osteopathic physicians apply manipulation in exactly the same manner, but each can point to cases in which he knows he has been instrumental in restoring health. My plea to young osteopathic physicians is not to overlook the advantages of osteopathic manipulative therapy in amenorrhea. The results achieved will be convincing proof of its effectiveness.

In addition I have found treatment to the musculoskeletal system quite useful. The patient sits lengthwise of the table with her feet on the table in front of her, knees flexed, arms hugging the knees, and head on the knees. In other words, the position is practically the universal one of flexion in utero. The doctor standing behind her uses either his elbows or the knuckles of clenched fists, one on either side of the spinous processes. Contact should be with the bare skin in order to avoid irritating the patient's skin from the rubbing of clothing. Beginning about the midthoracic spine, the physician slides his elbows or knuckles very slowly down through the lumbar area with a continuous deep pressure. This is quite painful at the time, but usually just one performance of this technic provides the patient with a feeling of well-being which, in my experience, is not obtained in any other manner. If pressure is kept deep and heavy, it has a marked effect on organs supplied by nerves from this area.

General measures, such as a well-rounded diet or, for the obese patient, a definite reducing program, are helpful. If the patient can lose 2 to 5 pounds per month for a time by diet, thyroid is administered only after her weight has reached a stationary level. Vitamin E will stimulate her vitality.

PROGNOSIS

The prognosis in amenorrhea depends primarily on the cause of the amenorrhea. If amenorrhea is the result of systemic disease, it will respond in direct proportion to the degree of success in treating the systemic cause. Successful treatment of the common cause usually results in successful treatment of the amenorrhea as well.

The prognosis is also influenced by the age of the patient. Although this paper deals mainly with amenorrhea of the adolescent, it should be mentioned that the

chances of restoring normal menstruation are much less in patients over 35 years of age. If the duration of amenorrhea is longer than a year, there is less chance of favorable results from treatment.

CONCLUSION

It was pointed out early in this paper that amenorrhea should be looked upon as a symptom. I have attempted to make this clear throughout the discussion. However, even though amenorrhea is a symptom, it is important to treat the patient as a whole, rather than the symptom alone.

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The Psychology of Delinquency and Vandalism in Juveniles*

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Frequently the doctor is the first to see the young vandal professionally. Parents, although they may have discussed the problem with school authorities, the minister, or legal authorities, may bring the child to the physician to learn if there is anything wrong with him. The physician also may be the first professional person to examine a child who shows incipient signs of vandalism.

In this paper I shall consider the psychology of the vandal, the dynamic factors which bring about his unsocial behavior, what it is that throws some children off balance, why they engage in wanton destruction of another's property, and what pushes them into this action when so many other channels are open to them.

No single cause of vandalism has been evidenced, though sometimes there are specific causes in certain geographic areas. The multiplicity of causes are in the areas of politics, economics, heredity, and environment; they are found in the parent, the teacher, the food, and the location of the home; they are said to include the war, the lack of a last frontier, and "keeping up with the Joneses"; they are all of these and more.

All of these causes relate to the patterning of the personality as the child grows from a completely dependent parasite at birth to a state of independence in adulthood. Most children mature through these years in a satisfactory manner. They learn to adjust to their living situations, to behave in a manner acceptable to society, and to be productive citizens. Most children learn to compensate for their own inadequacies and to sublimate their unacceptable urges or direct their desires which are not acceptable to society into adequate creative channels.

Relatively few become delinquent, and still fewer engage in vandalism. It is important to try to identify the psychology, the dynamics, and the motivating forces which lead to wanton destruction of property, no matter what the cause.

The fact is accepted that all individuals are different from each other, yet everyone has similar mechanisms and structures for maintaining life. Thus, each person may be said to have similar basic psychologic drives.

Kanner[1] brings out the significant thought that the struggle of the child for the gratification of needs is the basic drive in all behavior. Identification of the needs of all individuals is a problem. Various schools of thought describe needs in terms of their own theories of behavior and personality development. Ziskind[2] has described common needs that are recognized in all schools of thought and theories of behavior. They are variously noted as instincts, drives, wishes, desires, needs, urges, social forces, interests, and others. Such needs he lists as follows:

1. Physiologic needs or those biologic requisites of hunger and thirst which must be met for survival. The man who is hungry may eventually steal.

2. Safety needs, protecting the individual from injury or violence. These can be delineated as threats from physical disease and the fear of death in war. The feeling of insecurity may result from the fear of violence. When this fear is continuous, the person may strike out against the source of supposed violence or against the symbol of the source of violence.

3. The need for love. This may be thought of not only as love between sexes, but as acceptance, affection, and the feeling of belonging or of being part of a group. It is not a matter of whether or not the child is punished or how he is punished, but does he know he is loved; are adults consistent and fair in handling the child?

4. The need for self-esteem. It means "saving face." It is seen in "keeping up with the Joneses." The vandal meets this need among his fellows by doing a more destructive job than the others.

5. Creativity, the fifth basic need, which is recognized by all major schools of thought. It implies all forms of individual expression. If there is no provision for it in acceptable behavior, creativity is sought through unacceptable behavior.

Ziskind[2] lists these five basic needs in order of importance to the individual. Maslow[3] points out, how-

*Presented at the annual meeting of the American College of Osteopathic Pediatricians, Los Angeles, July 16, 1955.

ever, that any one specific drive or desire may be of greatest significance for a particular individual in his living situation. It is felt that when these basic motivating forces are not met adequately for the individual in his milieu, the resultant behavior is often pathologic and may be delinquent. In some cases the symptom is vandalism. In a given case, if the discovered causes of vandalism are investigated, it will be found that one or more of these needs is not being met adequately. The psychology of vandalism can therefore be thought of in terms of these desires and the way in which they are being met by the particular child.

In correcting the problem, the vandal's life may be evaluated in terms of these five basic urges. The value of this procedure cannot be overestimated in attempting to help patients, their parents, or the community.

Kanner[1] emphasizes the importance of dealing with the first act of delinquent behavior, no matter how insignificant it may seem. The physician, in taking a case history, may be the person who comes in contact with the child following his first unsocial activity, whether it is truancy, theft, or destruction.

Bertram Beck,[4] head of the special juvenile delinquency project of the United States Children's Bureau, has an outstanding approach to this problem. He believes that delinquency itself is not the big problem, but is only a symptom of the problem. He feels that the community must be alert for children with problems. Parents can worry too much about their children and not only "lose the kick of being parents" but create anxiety which is transmitted to the child.

Clendenen[5] has elaborated on the dynamics of the vandal, the child who is extremely hostile and acts out his rebellion against his family. All persons have some hostility, but the majority act it out in socially acceptable ways such as in playing golf, in "do-it-yourself" activities, and in playing ball. To the others, the church, the school building, or another's house may symbolize the parent or parent-figure, and the hostility is directed toward the symbol.

A paper on any aspect of the delinquency problem would be incomplete without mention of the Interim Report of the Subcommittee of the Committee on the Judiciary to Study Juvenile Delinquency in the United States.[6] The Committee reported that the number of children seen in juvenile courts increased from 300,000 in 1948 to 435,000 in 1953. Only 10 per cent of this increase can be attributed to the increase in juvenile population. When this rate is projected, it is anticipated there will be 785,000 in the juvenile courts in 1960. For every child brought before juvenile courts there are three offenders called to the attention of the police.

Narcotic addiction among young boys and girls, it was reported, is considered the most sinister of all problems. It was estimated that in New York City there are 7,500 juvenile addicts; in Los Angeles County 8 per cent of the children in juvenile court cases had contact with narcotics; in Denver 80 to 90 per cent of Spanish-American boys seen in juvenile courts had such contact; 250 children between the ages of 13 and 18 were addicted to drugs in Oklahoma City; and in Iowa 25 per cent of the girls in the state training school used marijuana habitually.[6]

Do these drugs help the child meet his basic needs? Does the transient elation from cocaine give him a feeling of self-esteem, of belonging? The later nervous discomfort, tension, and paranoid delusions are what make him a menace, a vandal. Vandalism can be stimulated by Benzedrine, Dexedrine, or mescaline. Does the case history provide information relative to the use of these drugs? During examination are symptoms identified which may result from the use of these drugs?

Drugs producing a depressant effect are used to relieve tension and pain; barbiturates and bromides are well-known examples. What basic need is met by relieving tension and gaining a feeling of well-being? The physician can answer this in terms of the individual child.

The Committee[6] indicated that because of the circumstances in which these drugs are obtained and used, a statistical study is difficult. However, it pointed out that not only do most adult addicts have a prior delinquent record but also that substantial figures were given to show the extent to which narcotics are used by delinquent children of both sexes.

Physicians must be alert to this problem, not only in administering the drugs but in recognizing symptoms of their use and in getting information from case histories. Psychiatric evaluation and treatment for tensions and conditions representing the apparent need for narcotics offer considerable help in limiting the use of such drugs in practice, thus avoiding possible addiction which may result in vandalism as an expression of unresolved problems.

The Senate report[6] identifies other problems such as inadequate recreation facilities in housing projects, the Armed Forces and the problem of young men with a record of difficulty with the law, juvenile employment problems, and pornographic material in relationship to delinquency. In it are discussed the school and the church in relation to these children and the importance of the community in its several aspects in solving the problem.

Comic books as they relate to the symptom of juvenile delinquency also have been studied in a special project.[7] It may be presumed that ideas pictured in comic books furnish another method of exciting the child to the vandal's way of establishing self-esteem, of belonging, and of creativity.

William C. Kvaraceus[8] has written that crime in the United States costs more than 15 billion dollars a year. A large proportion of this can be attributed to boys and girls in their teens. It would be subsumed that a goodly percentage of this cost must result from vandalism. The author states that much of the damage to property is not included in these figures since the incidents do not bring the child to court where statistics are compiled. He states that in Boston in 1953 the figures for school vandalism alone ranged from $53,752 to $76,811. In New York City in 1 year as many as 265,000 school windows have been the mark of vandals, while total school property damage reaches nearly a half-million dollars in the same period. It costs from $2,500 to $4,000 a year to keep a girl institutionalized for delinquency—as much as to send her to college.

Kvaraceus[8] outlines specific aids for every aspect of handling the delinquency problem. He points out the need for the community as a whole with all of its resources to work on the solution of the problem. He further points up the tenet of this paper, that delinquency or vandalism is a symptom of some problem lying within the community itself as well as within the child.

Kvaraceus identifies underlying psychologic aspects of the delinquent. He states that the behavior is an attempt of the child to meet some basic need. It is adjustive, in other words, rather than maladjustive. Because such behavior is troublesome, the child is brought into conflict with adults in the community. The need for status in the child reflects his family status, economically, socially, with regard to housing and schooling, and in relationship to group participation by parents.

There is overaggressiveness in the psychologic make-up of the delinquent, and I think we can assume the same is true of the vandal. The statement is made that society is apt to become preoccupied with the form of aggressiveness rather than the cause. Kvaraceus points out that aggressiveness and hostility result from feelings of frustration. Such causes may be poverty, rejection, insecurity, or being part of a minority group, as well as others previously mentioned. In terms of the five basic needs, it may be interpolated that frustration resulting in aggressiveness may stem from failure of the child to meet any or several of these urges.

The author[8] briefly discusses delinquency in terms of the freudian concept of meeting ego needs by impulsive acts. In terms of the basic needs already mentioned, and oversimplified, the id represents the basic urges necessary to survival; the ego represents the self or the conscious part of the personality; the superego is essentially the conscience or the "internalized" parent or parent-figure, the police in some cases. The vandal tries to make the id aspect of the personality "happy" by satisfying these urges, regardless of cost.

Other psychologists, as Kvaraceus points out, feel that interpersonal relations and the relationship with the environment are important factors in delinquency and, hence, in vandalism.

A study of the nutritional state and the body mechanics of the bony and soft tissues and the viscera of juvenile vandals might yield valuable information on the subject of vandalism. Nutrition and body mechanics are important in the osteopathic approach to any problem.

The basic osteopathic concept, which distinguishes the osteopathic school of practice, includes the principle of examining the total person in his total environment. This concept is basic in psychiatric thinking and practice. Osteopathic physicians caring for children are in a unique position to understand and take action regarding vandals.

The concept further states that, among other things, the individual is so designed that he can remain well or ward off or recover from noxious stimuli when he is maintained in an adequate environment, providing he inherits a stable and balanced organism.

This, too, is basic in the approach of almost everyone in the psychiatric field. Nature provided the mental mechanisms for adequate interpersonal relations and the ability to adjust to the present situation within a very wide range of activity. The child who is busy with constructive activity seldom appears in the courts. The child who learns the meaning of responsibility seldom is seen in juvenile institutions. The child who has the opportunity to discuss his problems with those who are interested and who have the time, or take the time, to listen and talk about them is seldom an offender. The child who has a stable religious experience in the home seldom is found among the vandals. The child who is nourished adequately, who is secure in the knowledge that he is safe, who knows he is loved and is a real part of his family and community whether it is the school, the church, or other groups, who realizes self-esteem, and who has adequate and appropriate direction for his creativity is not seen in the courts and is not found destroying the property of others without due cause. The child who has a stable inheritance, a mental capacity which enables him to evaluate and to learn, who is mechanically adequate, and who is guided to emotional maturity so that he can face reality is not seen in the child guidance clinic.

In conclusion, the psychologic aspects of vandalism are many. They result from the reaction of children to attitudes. Those closest to the child, with him first, and representing the greatest force are the parents. But he is in school 6 or 7 hours daily from the time he is 6 years of age. He is part of the church, of the theatre, of the grocery store; he walks on the streets, plays with his neighbors, looks at television, listens to the radio, reads, wears clothes, eats, and sleeps.

The physician as a part of the community might well ask himself what his responsibilities are in the problem of vandalism. Does he talk about this problem constructively with patients, parents, and religious leaders? Not only is the physician working with parents and children, not only does he counsel with regard to parent and teacher attitudes, not only does he counsel the child, but he is considered a leader by the community. Is he taking advantage of this position by stimulating adequate community attitudes and contributing to the development of stable, useful, happy young citizens? Is he taking positive action because he is thinking positively?

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Journal A.O.A.
Vol. 55, No. 4

Pulmonary Hyaline Membrane

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The diagnosis of pulmonary hyaline membrane is all too often made on the autopsy table. In view of its high incidence as a cause of death within the first 48 hours in premature and postmature infants, babies of diabetic mothers, and those delivered by cesarean section, it is important for physicians to familiarize themselves with this pathologic entity and the clinical syndrome that is part of it. At a recent symposium[1] dealing with this disease it was the consensus that a correct antemortem diagnosis of pulmonary hyaline membrane could be made in any infant weighing more than 1,800 grams, and one physician,[2] referring to children of diabetic mothers, stated that his "batting average" was nine out of ten.

At the Chicago Lying-In Hospital between 1948 and 1949, this condition was the only pathologic change found at postmortem examination in 40 per cent of all infants weighing between 1,000 and 2,500 grams and was the leading cause of death of live-born infants.[3] Among infants weighing over 2,500 grams it was proportionately more common among those delivered by cesarean section. If the deaths due to erythroblastosis and malformations were eliminated from that series, it became almost the only cause of death in infants delivered by cesarean section in that hospital.

Typically at delivery there is an uneventful initiation of respirations, and the baby quickly assumes the pink color of adequate oxygenation. Less frequently there may be initial cyanosis which is refractory to the administration of oxygen. There is then a latent period, generally from 1 to 6 hours, during which the infant appears normal. At this time the infant starts to show evidence of distress. Progressive cyanosis and dyspnea appear, and the baby seems to be making tremendous efforts at inspiration as though he were trying to overcome some severe obstruction in the bronchopulmonary tree. All the accessory muscles of respiration are brought into play. There is lower sternal retraction from a strong diaphragmatic tug, which becomes progressively deeper as the condition advances. The ribs retract and a sucking in of the supraclavicular spaces can be seen. Initially, and for a variable period, the inspiratory effort seems to be labored, and then the baby seems to have trouble expiring the air from the chest. It has been observed that the knee-chest position seems to ease the expiratory difficulty.[4] The diaphragmatic action resulting from the tug disappears and is replaced by an "undercurling" of the thoracic cage which occurs because of the now tremendous excursions of the upper chest which is trying to compensate for the loss of diaphragmatic action. This loss of diaphragmatic function has been confirmed by pneumotracheographic studies and is considered a poor prognostic sign.[4]

During the latent period between birth and the onset of symptoms, percussion and auscultatory findings are within normal limits, but with the onset of distress these findings become extremely variable, with the picture changing from one hour to the next. Percussion dullness and the absence of breath sounds may be demonstrated in a given area, and an hour later the same area may be hyper-resonant. Death usually takes place in from 8 to 30 hours as a result of exhaustion and the inability to obtain enough oxygen to maintain life.

X-ray studies are not typically diagnostic; however, a granular, fine hyaline appearance of the lung fields has been reported with some consistency. Radiologic evidence of massive atelectasis is not consistent with the diagnosis.

If the infant survives, a 48-hour period pneumonia is usually superimposed, with or without a response to infection. Death does not inevitably occur. Cases with less extensive involvement, given proper care, will clear up, usually within 5 days.

At autopsy, the pathologic findings in the lungs are quite striking.[5] The lungs are the size of well-expanded lungs but they have the consistency of liver. Instead of being the yellow-pink of lungs containing air or the light violet of nonexpanded fetal lungs, they are uniformly dark reddish purple. If the infant survived a 48-hour period, a superimposed inflammatory state is present characterized by a granular consistency. Ordinarily, however, the lungs are of uniform consistency. The whole lung or individual portions sink when placed in water despite the fact that a little air always remains in the proximal portion of the pulmonary tree.

Microscopic examination reveals widespread resorption of air, and the walls of many alveolar ducts and most of the alveoli are collapsed, giving a solid appearance to the lung tissue between the few alveoli that remain open. Great difficulty is experienced when trying to expand the lungs by injecting air, the proximal alveoli becoming greatly distended while the distal alveoli become even more collapsed. However, if fluid is injected, the atelectatic alveoli are easily opened and microscopic inspection soon shows that the lungs are normally developed.

Intense capillary engorgement is responsible for the color and increase in weight and is one of the most striking findings on histologic examination.

The classical pulmonary hyaline membrane is seen on the inner surface of the bronchioles and alveolar ducts that remain open. It appears as an irregular layer of homogeneous to granular acidophilic material extending into the bronchioles and alveolar ducts. It forms a mechanical barrier to normal respiratory exchange by blocking off some portions of pulmonary tissue and by coating the remainder in such a way that the capillaries are deprived of contact with the oxygen-containing atmosphere. The hyaline membrane directly overlies the intact epithelium of the air spaces, the respiratory cells themselves not being incorporated within the outside limits of the membrane. Squamous cells are sometimes seen lying between the membrane and the walls of the alveolar duct.

Histochemically, the membranes seem to contain a protein which consists of tyrosine and arginine. A carbohydrate moiety is attached to the protein. Fatty substances in small amounts are present and are probably devoid of hyaluronic acid.[6]

Investigators agree in describing the clinical and pathologic findings in this disease, but there is much disagreement as to the origin and pathogenesis of the pulmonary hyaline membrane. The prevailing hypothesis, which is by no means a consensus, is as follows:

In the process of physiologic intrauterine fetal respirations, a tidal flow of amniotic fluid takes place between the amniotic sac and the upper respiratory passages of the fetus. With normal fetal respiratory excursions, which are shallow, the lower part of the respiratory tract is not reached by the amniotic fluid. However, when fetal anoxia occurs, from any one of a variety of causes, gasping respirations occur and the protein-laden amniotic fluid is drawn into the terminal bronchioles, alveolar ducts, and sometimes the alveoli themselves. The infant is delivered prematurely if the condition causing fetal anoxia precipitates delivery or necessitates the induction of labor. After birth, as a result of extrauterine respiration of at least 1 to 2 hours' duration, this material (protein, squamous cells, occasional vernix) is dispersed within the respiratory bronchioles, alveolar ducts, and alveoli.

As absorption of the fluid element of the amniotic contents takes place, there is concentration of its aforementioned solid elements, and a membrane is formed which seals off the openings of the bronchioles, alveolar ducts, and alveoli, thereby blocking an effective gaseous exchange. If the blocked alveolus had formerly been expanded by air, the air is now absorbed and the involved area becomes secondarily atelectatic. If the alveolus had not yet been opened by air, it remains in its state of primary atelectasis.

The above hypothesis is based on the results of experimental work done in this field. Its integrity must of course be tested by proving whether or not such phenomena as effective intrauterine fetal respirations and tidal flow of anniotic fluid do exist.

Snyder and Rosenfeld[7] observed and illustrated rhythmic respiratory movements of fetal rabbits within their amniotic membranes and showed that continuity existed between the amniotic sacs and fetal lungs by injecting India ink into the amniotic sac 20 minutes before the animal was sacrificed. Entrance of the stained amniotic fluid into the fetal lung was evidenced by the completely stained condition of all parts of the lung. An apneic fetus subjected to the same procedure presented unstained lungs. This would seem to indicate that fetal respirations and aspiration of amniotic fluid occur in normal intrauterine respiration in the rabbit fetus.

In attempting to show that aspiration of amniotic fluid was necessary to normal development of the fetal lung, Potter[3] injected Thorotrast into the amniotic sac of small human fetuses 12 to 24 hours prior to delivery and demonstrated radiologically and on microscopic section the presence of Thorotrast in the lung fields and throughout the gastrointestinal tract. If the material was injected just prior to delivery it was not visualized. She concluded that a certain amount of time was necessary for the tidal flow of amniotic fluid to leave the particles of Thorotrast to concentrate within the lung.

However, Gruenwald[8] suggests that concentration of Thorotrast occurs when the amniotic fluid is absorbed by the lungs, leaving the Thorotrast behind the capillary membrane at the alveolar wall. This was further borne out by demonstration that rabbit fetuses could swim in saline baths until they stopped breathing and could then be revived after aspirating a large amount of the saline. With this work in mind it may be reasonable to assume that what actually occurs is an inflow of amniotic fluid without an appreciable outflow, rather than a tidal flow of fluid between the lungs and amniotic sac.

Another pertinent question is whether intrauterine fetal anoxia causes increased respiratory excursions with aspiration of alveolar debris and amniotic fluid into the bronchioles and alveolar ducts. On the basis of available evidence, this question must remain unanswered.

While it is agreed that the pulmonary hyaline membrane is derived from protein, there is disagreement as to the source of the protein. Amniotic fluid has a relatively low protein content, and a large amount of fluid would be necessary to form the membranes that are found. Moreover, pulmonary hyaline membranes found in children and adults are seen in conditions such as arteritis, x-ray pneumonitis, and pneumonitis of rheumatic fever in which there is vascular damage. This would indicate that a mechanism exists for blood proteins to escape from an injured capillary and travel the infinitesimal distance to the alveolar duct. Since premature infants are prone to vascular fragility, the source of the protein may very well be the blood rather than the amniotic fluid. Blood as the sole source of the membrane would not account for the amniotic debris which is found in the membrane.

Possibly, therefore, the theory of fetal anoxia explains the entire picture. It may very well be that fetal anoxia is responsible for the capillary damage which releases the protein, for the gasping fetal respiration, the consequent deep aspiration of amniotic fluid, and the settling out of the amniotic debris which is part of the membrane.

This explanation would seem to be well borne out by the recent work of Gitlin[9] on the nature of the membrane. Using a fluorescein tag, he was able to produce a fluorescent fibrin membrane in edematous lungs, in which the pulmonary edema fluid was combined with amniotic fluid. Finding fibrinogen in the edema fluid and thromboplastin in the amniotic fluid, Gitlin then explains the formation of the pulmonary hyaline membrane by the union of these fluid components when the edema fluid of damaged lungs is combined with amniotic fluid which is deeply aspirated by a hypoxic fetus.

It has been shown that vagal influences may operate in the production of the hyaline membrane. Miller and his associates[10] were able to produce membranes in twenty rabbits on which vagotomy at the midcervical level had been performed. These membranes were similar to those found in premature babies, and the longer the period of life after the procedure, the greater was the amount of hyaline membrane found. The time of appearance of the membranes after vagotomy approximated closely the time of appearance of the membranes in newborn infants in respect to the time of birth. Both membranes contained polysaccharide aldehydes. The similarity of the lesions produced in vagotomized rabbits to the lesions found in premature infants suggests that there may be factors common to both which are responsible for these pulmonary changes. As to the origin of the membrane, an alveolar transudate or pulmonary edema was found in association with membranes produced in the vagotomized rabbits that was similar to edema that his group found in their series of premature humans.

That premature infants have poor vagal tone is evidenced by tachycardia, irregular respirations, and a weak gag and cough reflex. Experimental membranes have been produced by the intratracheal injection of dilute hydrochloric acid in laboratory animals, and this could conceivably occur in premature babies who aspirate their gastric contents.

The role of cesarean section is a special one in this condition because of the great incidence of deaths of newborns weighing over 2,500 grams delivered by section. Approximately 50 per cent of deaths occurring after section in infants born alive show this pathologic state as the cause of death. Why pulmonary hyaline membrane should occur so frequently with this method of delivery is again a matter of conjecture. In some cases the maternal indication for cesarean section may also produce fetal anoxia, thereby setting the stage for formation of the membrane. Delivery by section is unphysiologic and may be more likely to promote the formation of hyaline membranes in the lungs of newborn infants than does delivery from below.

When the uterus is opened and the membranes of the amniotic sac are ruptured, the infant is seen lying in a pool of amniotic fluid. During extrication of the baby, its body is exposed to and chilled by the lower room temperature, and it has been observed to gasp or even cry while its head is still submerged in the amniotic fluid within the uterus. Furthermore, with this method of delivery the chest is not subjected to the wringer-like action of the birth canal. Copious amounts of clear bubbly fluid issue from the nose and mouth of the infant born by cesarean section for the first few hours of its life. Finally, anesthesia, operative manipulation, and absence of uterine contractions must all be considered in evaluating the part played by cesarean section in the pathogenesis of pulmonary hyaline membrane.

In the absence of a known specific cause, it is understandable that a specific treatment does not exist. However, some general measures directed toward the prevention and treatment of pulmonary hyaline membrane have been utilized with some degree of success.

The physician should anticipate, try to prevent, and be prepared to treat this pathologic state when faced with a premature delivery, a cesarean section, or the delivery of a baby of a diabetic mother. Prenatal prevention concerns itself with measures taken to avoid prolonged intrauterine anoxia and, whenever possible, avoidance of cesarean section. When section is indicated it is probably wise to extricate the infant from the uterus as rapidly as possible.

Whatever method of delivery is used, the airway must be closed off until the nose, mouth, and throat can be aspirated with a bulb syringe or DeLee aspirator. If the infant requires resuscitation, direct laryngoscopy by a skilled operator is utilized, and a tracheal catheter is inserted for aspiration and oxygenation. To avoid aspiration of gastric contents, it is advisable to empty the stomach by means of an appropriately sized rubber catheter. This is particularly indicated for babies delivered by section who have a copious amount of gastric and pulmonary fluid.

Since it is the consensus of the pathologists that no material exists in the larger bronchi that could be removed by bronchoscopy, this procedure is needless, and since deaths have been reported immediately following it, in my opinion bronchoscopy is absolutely contraindicated. The fluid within the larger bronchi can easily be removed by adequate postural drainage.

It is obvious from their cyanosis that these infants are suffering from lack of oxygen. A great many of them become cyanotic when they are removed from oxygen, but their color remains quite good when they are maintained in a high oxygen environment. It is thought that a supersaturated oxygen or oxygen mist atmosphere of 90 to 95 per cent humidity is particularly beneficial in this condition. Some workers have simply used a spray of water, while others utilized detergent solutions such as Alevaire or protein-splitting enzyme substances such as Tryptar or streptokinase-streptodornase. Gellis[2] routinely placed all infants delivered by cesarean section in mist for a 24-hour period immediately following delivery, and he feels that this may prevent or minimize the condition.

It has been my practice to use detergent mist routinely for all premature babies, babies delivered by section, and babies of diabetic mothers. Since respiratory distress starts within the first 24 hours of life, these babies are removed from this environment if they are normal at the end of that period.

Adequate postural drainage, except when intracranial hemorrhage is suspected, should be instituted immediately, and the incubator should be arranged so that this position can be maintained for the first 24 hours.

Since superimposed pulmonary infection is noted to follow in 48 hours, these infants should be protected by parenteral administration of penicillin and streptomycin for a 3-day period.

Feeding by mouth should be withheld until all signs of respiratory distress have abated. Parenteral fluids may be administered, depending upon the degree of hydration.

If the extent of involvement is not overwhelming and if careful attention is given to prophylaxis and symptomatic treatment, the very high mortality rate that now exists for this disorder of pulmonary ventilation may be lowered.

SUMMARY

Pulmonary hyaline membrane causing abnormal pulmonary ventilation is a leading cause of death in premature infants, newborns of diabetic mothers, and those newborns delivered by cesarean section. It is an acidophilic material consisting largely of fibrin which lines the inner surfaces of the bronchioles and alveolar ducts and forms a mechanical barrier to physiologic respiratory exchange.

A latent period of from 1 to 6 hours usually occurs between an apparently uneventful delivery in which the newborn appears well and the onset of apparent respiratory distress.

Several theories explaining the etiology of pulmonary hyaline membrane have been presented and discussed. The possible occurrence of this condition should be anticipated in any birth involving a premature infant, a cesarean section, or a diabetic mother, and proper prophylactic measures should be undertaken.

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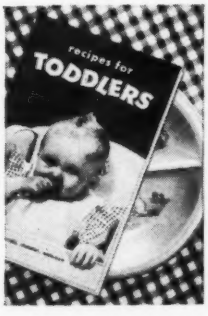

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Conventions and Meetings

Announcements

American Osteopathic Association, Sixtieth Annual Convention, Hotel Statler, New York City, July 16-20, 1956. Program Chairman, Myron C. Beal, 17 Vick Park A., Rochester 7, N. Y.

Academy of Applied Osteopathy, annual meeting, Hotel Waldorf, New York City, July 20. Secretary, Margaret W. Barnes, Box 1345, Carmel, Calif.

American College of Osteopathic Obstetricians and Gynecologists, annual meeting, Neil House Hotel, Columbus, Ohio, February 6-9. Program Chairman, Richard E. Eby, 1247 N. Park Ave., Pomona, Calif. Secretary, Harold K. Morgan, 3268 W. 32nd Ave., Denver 11.

American Osteopathic College of Proctology, annual meeting, Hotel Statler, Dallas, Tex., March 27. Secretary, John W. Orman, 1623 E. 15th St., Tulsa 14, Okla.

American Osteopathic Society of Proctology, annual meeting, Hotel Statler, Dallas, Tex., March 28-30. Program Chairman, E. R. Horton, Jr., 214 E. DeWald St., Fort Wayne 5, Ind. Secretary, Carl S. Stillman, Jr., 3523 Fifth Ave., San Diego 3, Calif.

British Columbia: See Northwest Osteopathic Convention.

California, annual meeting, Mission Inn, Riverside, May 24-27. Program Chairman, Milton Futterman, 2028 E. First St., Los Angeles 22. Executive Secretary, Mr. Thomas C. Schumacher, 1298 Wilshire Blvd., Los Angeles.

Colorado, annual meeting, Denver, May 5. Program Chairman, A. E. Nichols, 1332 Del Mar Parkway, Denver 8. Secretary, C. Robert Starks, 1459 Ogden St., Denver 18.

Connecticut, midyear meeting, February 18-19. Program Chairman, E. R. Lamb, 44 Whitney St., Hartford 5. Secretary, C. W. Cornbrooks, 182 Post Road, Darien.

Eastern Osteopathic Association, annual meeting, Hotel Statler, New York City, March 24-25. Program Chairman, Chester D. Losee, 212 Prospect St., Westfield, N. J. Secretary, Frank B. Tompkins, Baltimore Life Bldg., Baltimore 1.

Florida, annual meeting, Fort Harrison Hotel, Clearwater, May 17-19. Program Chairman, Paul E. Wilson, 802 E. Ocklawaha Ave., Ocala. Secretary, Dominic Raffa, 5009 Central Ave., Tampa 3.

Georgia, annual meeting, General Oglethorpe Hotel, Savannah, April 26-28. Program Chairman, Wade Holloway, 509 Upchurch Bldg., Thomasville. Secretary, Walter B. Elliott, Jr., 702 Mortgage Guarantee Bldg., Atlanta 3.

Idaho: See Northwest Osteopathic Convention.

Indiana, annual meeting, Notre Dame University and Morris Inn, South Bend, May 14-16. Program Chairman, A. F. Kull, South Bend Osteopathic Hospital, 118 S. William St., South Bend 2. Secretary, Arabelle Baker Wolf, 809-13 Odd Fellows Bldg., Indianapolis 4.

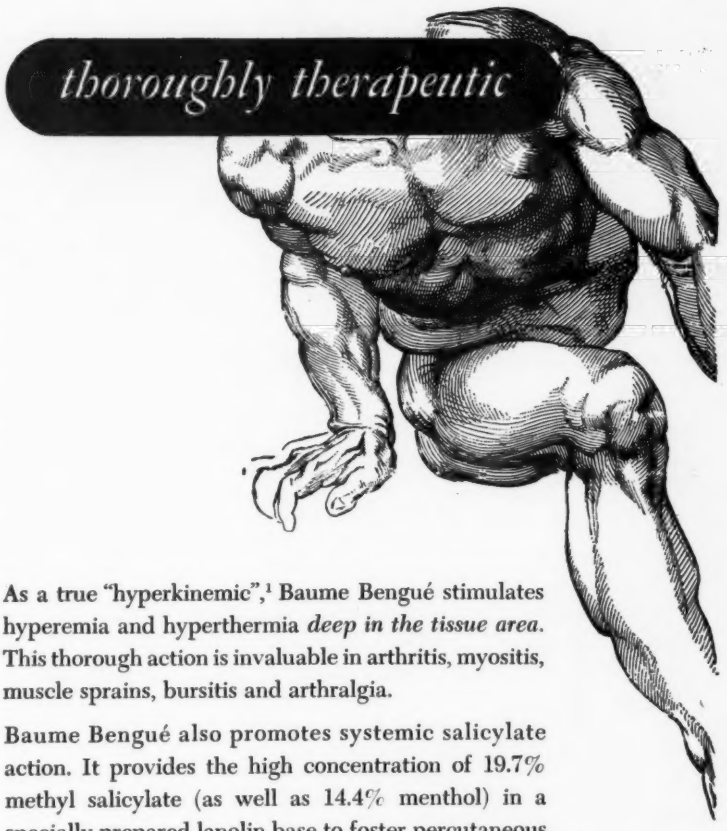
Iowa, annual meeting, Hotel Savery, Des Moines, May 20-22. Program Chairman, Clive R. Ayers, Grant. Secretary, Mr. Herman A. Walter, 200 Walnut Bldg., Des Moines 9.

Maine, annual meeting, Samoset Hotel, Rockland, June 14-16. Professional Education Chairman, Richard Wallace, Hollis Center. Secretary, Roswell P. Bates, 72 Main St., Orono.

Massachusetts, annual meeting, Hotel Somerset, Boston, January 21-22. Program Chairman, Everett L. Pierce, Pleasant St., East Dennis. Executive Secretary, Mrs. Gladys M. Stockdale, 524 California St., Newtonville.

Minnesota, annual meeting, Minneapolis, May 3-5. Program Chairman, Constance Idtse, 47 S. Ninth St., Minneapolis 2. Secretary, E. R. Komarek, 301 Granite Exchange Bldg., St. Cloud.

National Osteopathic Child's Health Con-



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New Jersey, annual meeting, Essex House, Newark, March 10-11. Program Cochairmen, George W. Northup, 104 S. Livingston Ave., Livingston, and Harry A. Sweeney, 25 S. Illinois Ave., Atlantic City. Executive Secretary, Mr. I. J. Tecker, 21 Hancock St., Riverside.

Northwest Osteopathic Convention, Chinook Hotel, Yakima, Washington, June 25-27. Program Chairman, Wilbert B. Saunders, 4730 University Way, Seattle 5.

Ohio, annual meeting, Neil House, Columbus, May 7-9. Program Chairman, George O. Hoover, 91 S. Main St., Oberlin. Executive Secretary, Mr. William S. Konold, 50 E. Broad St., Columbus 15.

Ontario, annual meeting, Royal Hotel, Guelph, May 3-5. Secretary, A. V. DeJardine, 205 Yonge St., Toronto 1.

Oregon: See Northwest Osteopathic Convention.

Rhode Island, annual meeting, Warwick Country Club, Warwick, March 7-8. Program Chairman, Richard Dowling, 1039 Broad St., Providence 5. Secretary, J. Weston Abar, 824 Broad St., Providence 7.

Texas, annual meeting, Baker Hotel, Dallas, April 22-28. Program Chairman, Robert J. Brune, 1819 S. Brownlee, Corpus Christi. Executive Secretary, Phil R. Russell, 512 Bailey St., Fort Worth 7.

Utah, annual meeting, Ambassador Athletic Club, Salt Lake City, June 23. Program Chairman, Wilford G. Hale, 506 West 2nd South, Logan. Secretary, Alice E. Houghton, 600 Zion's Savings Bank Bldg., Salt Lake City 11.

Virginia, annual meeting, The Lodge, Williamsburg, May 25-26. Program Chairman, Vincent H. Ober, 407-11 Bankers Trust Bldg., Norfolk 10. Secretary, John A. Cifala, 2778 N. Washington Blvd., Arlington.

Washington: See Northwest Osteopathic Convention.

West Virginia, annual meeting, Daniel Boone Hotel, Charleston, June 2-5. Program Chairman, Walter B. Goff, 429 16th Street, Dunbar. Secretary, Guy E. Morris, 542 Empire Bank Bldg., Clarksburg.

Western States Osteopathic Society of Proctology, annual meeting, Mayo Hotel, Tulsa, Okla., October 8-10. Program Chairman, Glenn Miller, 2100 N. Robinson St., Oklahoma City 3. Secretary, Earle F. Waters, 925 E. South Temple St., Salt Lake City 2.

Wisconsin, annual meeting, Hotel Wisconsin, Milwaukee, April 23-25. Program Chairman, R. D. Walling, 709 Oak St., Baraboo. Secretary, Edwin J. Elton, 1518 N. 70th St., Wauwatosa 13.

State and National Boards

ARIZONA

Those interested in professional examinations should contact Russell Peterson, D.O., secretary, Osteopathic Board of Registration and Examination in Medicine and Surgery, 2747 East McDowell Road, Phoenix.

Basic science examinations March 20 at the University of Arizona, Tucson. Applications must be filed 2 weeks prior to the examination. Address Herbert D. Rhodes, Ph.D., secretary, Basic Science Board, University of Arizona, Tucson.

COLORADO

Basic science examinations in March at the Y.M.C.A. Bldg., E. 16th Ave. and Lincoln St., Denver. Address Esther B. Starks, D.O., secretary, Basic Science Board, 1459 Ogden St., Denver 18.

CONNECTICUT

Basic science examinations February 11 in Room 23, Lampson Hall, Yale University, New Haven. Applications must be filed 2 weeks prior to examination. Address Miss M. G. Reynolds, executive assistant, Board of Healing Arts, 52 Whitney Ave., New Haven 10.

DELAWARE

Examinations January 10-12. Address Joseph S. McDaniel, M.D., secretary, Board of Medical Examiners, 229 S. State St., Dover.

ILLINOIS

Examinations in January in Chicago. Address Mr. Frederic B. Selcke, Superintendent of Registration, Department of Registration and Education, State House, Springfield.

IOWA

Basic science examinations January 10 at the Capitol Bldg., Des Moines. Address Ben H. Peterson, Ph.D., secretary, Board of Basic Science Examiners, Coe College, Cedar Rapids.

KANSAS

Examinations February 16-18 at Topeka. Address Forrest H. Kendall, D.O., secretary, Board of Osteopathic Examination and Registration, 420½ Pennsylvania Ave., Holton.

MASSACHUSETTS

Examinations January 10. Address Robert C. Cochrane, M.D., secretary, Board of Registration in Medicine, Room 37, State House, Boston 33.

MICHIGAN

Basic science examinations February 10-11 at Detroit and Ann Arbor. Applications must be filed prior to February 1. Address Mrs. Anne Baker, secretary, Board of Examiners in the Basic Sciences, 116 Mason Bldg., Lansing.

MINNESOTA

Eugene C. Herzog, Brainerd, has been reappointed to the Board of Osteopathic Examiners for a term of 5 years.

MONTANA

Examinations March 6. Address Asa Willard, D.O., secretary, Board of Osteopathic Examiners, Wilma Bldg., Missoula.

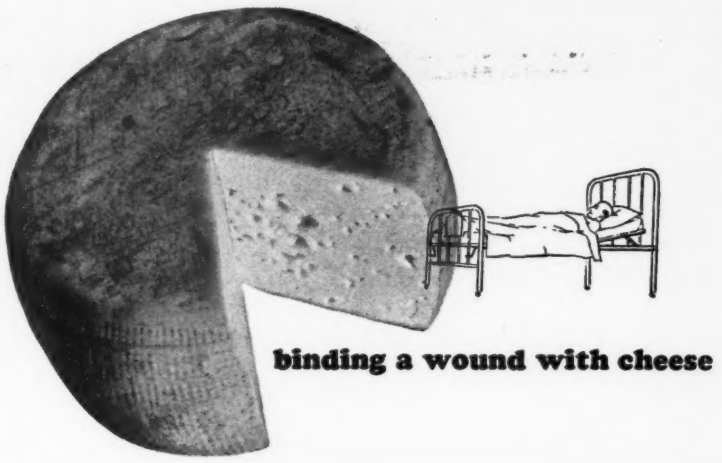
NEBRASKA

Basic science examinations January 10-11. Applications must be filed 15 days prior to examination. Address Mr. Husted K. Watson, Director, Bureau of Examining Boards, Department of Health, State Capitol Bldg., Lincoln 9.

NEVADA

Professional examinations January 27 at 210 W. Second St., Reno. Applications must be filed by January 13. Address Walter J. Walker, D.O., secretary, Board of Osteopathic Examiners, 210 W. Second St., Reno.

Basic science examinations January 3. Address Donald G. Cooney, Ph.D., secretary, Board of Examiners in the Basic Sciences, Box 9005, University Station, Reno.



A wound is as strong as the connective tissue that holds it together... and the maximum strength of a wound is reached more rapidly when the diet contains liberal amounts of protein for growth of connective tissue. [1]

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1. Baborka, C. J.: Treatment by Diet, ed. 5, Philadelphia, J. B. Lippincott Company, 1948, p. 607.
2. Sebrell, W. H., in Stieglitz, E. J.: Geriatric Medicine, ed. 2, Philadelphia, W. B. Saunders Company, 1949, p. 194.
3. Morgan, D. B.: J. Missouri M. A. 49:896 (Nov.) 1952.

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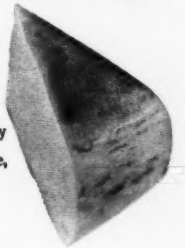


NEW HAMPSHIRE

Examinations March 14-15 at Concord. Address John S. Wheeler, M.D., secretary, Board of Registration in Medicine, State House, Concord.

NEW MEXICO

Basic science examinations January 15. Address Mrs. Marguerite Cantrell, secretary, Board of Examiners in the Basic Sciences, P.O. Box 1522, Santa Fe.

NEW YORK

Examinations February 7-10. Address John W. Paige, M.D., chief, Bureau of Professional Examinations and Registrations, 23 S. Pearl St., Albany 7.

NORTH DAKOTA

Examinations January 14 at Minot. Applications must be filed by December 15. Address G. L. Hamilton, D.O., secretary, Board of Osteopathic Examiners, Ringo Bldg., 119 S. Main St., Minot.

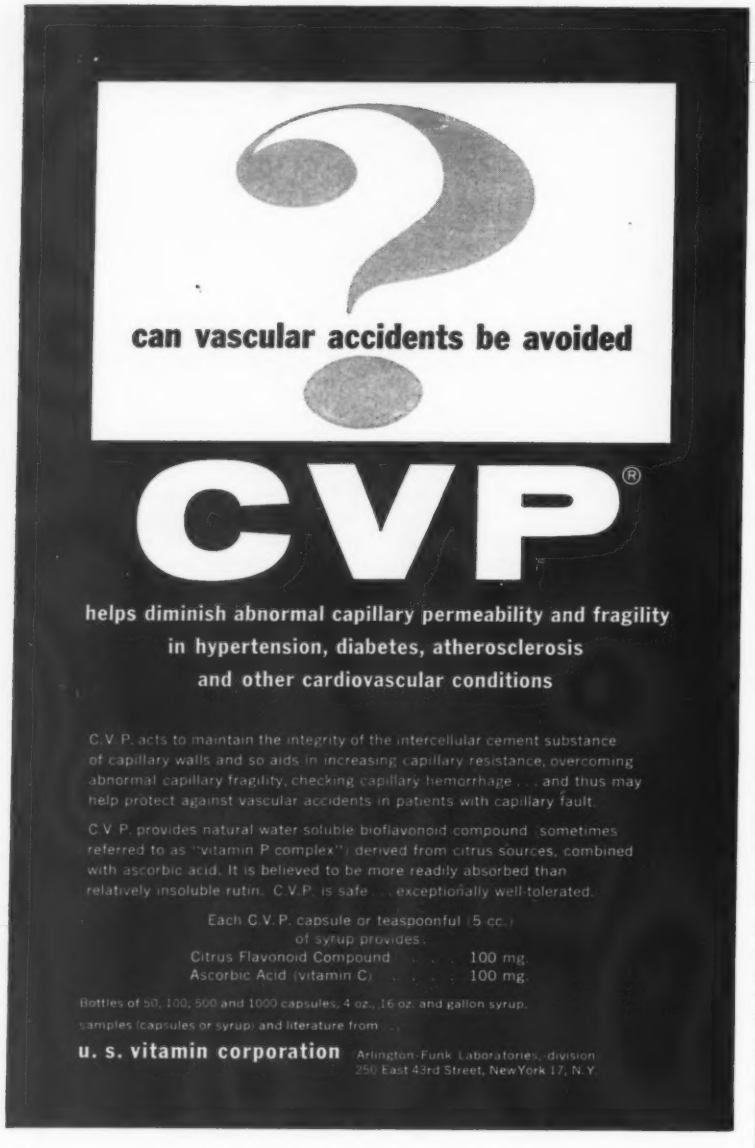
J. O. Thoreson, Mandan, has been reappointed to the Board for a term expiring July 1, 1958.

OREGON

Reciprocity applicants will be interviewed at the regular meeting of the Board of Medical Examiners, January 14-15. Address Mr. Howard I. Bobbitt, executive secretary, 609 Failing Bldg., Portland 4.

RHODE ISLAND

Professional examinations January 5-6. Address Mr. Thomas B. Casey, Ad-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ministrator of Professional Regulation, 366 State Office Bldg., Providence.

Basic science examinations February 15-16 in Room 366, State Office Bldg., Providence. Applications must be filed by January 26. Address Mr. Casey.

SOUTH DAKOTA

Professional examinations January 17-18 at Sioux Falls. Address Mr. John C. Foster, executive secretary, Board of Medical and Osteopathic Examiners, Room 300, First National Bank Bldg., Sioux Falls.

TENNESSEE

Examinations February 8 at Nashville. Applications must be filed by January 15. Address M. E. Coy, D.O., secretary, Board of Examination and Registration for Osteopathic Physicians, 1226 Highland, Jackson.

VERMONT

Examinations January 25-26 at the State House, Montpelier. Applications must be filed by January 15. Address Charles D. Beale, D.O., secretary, Board of Osteopathic Examination and Registration, Mead Bldg., Rutland.

WASHINGTON

Professional examinations in January. Address Mr. Edward C. Dohm, secretary, Professional Division, Department of Licenses, Olympia.

Basic science examinations in January. Applications must be filed 30 days prior to examination. Address Mr. Dohm.

WISCONSIN

Professional examinations January 10 at Madison. Address Thomas W. Tormey, Jr., M.D., secretary, Board of Medical Examiners, State Office Bldg., 1 West Wilson St., Madison.

WYOMING

Examinations February 6 at Cheyenne. Address Franklin D. Yoder, M.D., secretary, Board of Medical Examiners, New State Office Bldg., Cheyenne.

REREGISTRATION OF OSTEOPATHIC LICENSES

December 31—Tennessee, $5.00. Address M. E. Coy, D.O., secretary, Board of Examination and Registration for Osteopathic Physicians, 1226 Highland, Jackson.

Prior to January 1—Arizona, not more than $10.00. Address Russell Peterson, D.O., secretary, Osteopathic Board of Registration and Examination in Medicine and Surgery, 2747 E. McDowell Rd., Phoenix 22.

January 1—California, $20.00 for residents and nonresidents. Address Glen D. Cayler, D.O., secretary, Board of Osteopathic Examiners, 1013 Forum Bldg., Sacramento 14.

January 1—Florida, $5.00. Address W. S. Horn, D.O., secretary, Board of Osteopathic Medical Examiners, 1500 Eighth Ave., Palmetto.

January 1—Maine, $4.00. Address George F. Noel, D.O., secretary, Board of Osteopathic Examination and Registration, Monument Square, Dover-Foxcroft.

January 1—Manitoba, $5.00. Address W. Kurth, D.O., secretary, Board of Osteopathic Physicians, 248 Moorgate Blvd., Deer Lodge, Winnipeg.

January 1—New York, $5.00, biennially. A physician receiving a license the second year of any biennial registration period pays a fee of $2.50 for a certificate expiring December 31 of such second year. Address Stiles D. Ezell, M.D., secretary, Bureau of Professional Examinations and Registrations, 23 S. Pearl St., Albany 7.

January 1—Ontario, $35.00. Address D. Gordon Campbell, D.O., secretary, Board of Directors of Osteopathy, 2 Bloor St., E., Toronto 5.

January 1—Pennsylvania, Surgeons Examining Board, $10.00. Address Mrs. Katherine M. Wollet, acting secretary, Board of Osteopathic Examiners, Bureau of Professional Licensing, Harrisburg.

January 1—Texas, $5.00. Address M. H. Crabb, M.D., secretary, Board of Medical Examiners, 1714 Medical Arts Bldg., Fort Worth 2.

January 1—Utah, $3.00. Address Alice E. Houghton, D.O., secretary, Osteopathic Examining Board, 600 Zion's Savings Bank Bldg., Salt Lake City 1.

January—Alberta. No registration. Pay $10.00 a year membership in the College of Physicians and Surgeons, Alberta, in January.

During January—Connecticut, $2.00. Address Frank Poglitsch, D.O., secretary, Osteopathic Examining Board, 300 Main St., New Britain.

During January—Minnesota, $2.00. Address Wallace F. Kreighbaum, D.O., secretary, Board of Osteopathic Examiners, 2748 Hennepin Ave., Minneapolis 8.

During January—Wisconsin, $3.00. Address Thomas W. Tormey, Jr., M.D., secretary, Board of Medical Examiners, State Office Bldg., 1 W. Wilson St., Madison.

January 31—British Columbia, amount of fee set at Annual Meeting of Council of College of Physicians and Surgeons of British Columbia. Address Lynn Gunn, M.D., Registrar, College of Physicians and Surgeons of British Columbia, 1807 W. 10th Ave., Vancouver 9.

Before February 1—Vermont, $3.00 for residents, $2.00 for nonresidents. Address Charles D. Beale, D.O., secretary, Board of Osteopathic Examination and Registration, Mead Bldg., Rutland.

Before March 1—Colorado, $2.00 if legal resident of Colorado; $10.00 if not legal resident of Colorado. Address Mrs. Beulah H. Hudgens, executive secretary, Board of Medical Examiners, 831 Republic Bldg., Denver 2.

EXAMINATION BY NATIONAL BOARD

The National Board of Examiners for Osteopathic Physicians and Surgeons conducts Parts I and II of its examination on the first Thursday and Friday of each May and December at the six approved colleges. Application blanks may be obtained from the secretary or the dean of the college, and the completed application blank, together with a passport photograph and check for the parts to be taken, must be in the secretary's office by the November 1 or April 1 preceding the examination.

Examinations in Part 1 consist of anatomy, including histology and embryology; physiology; physiological chemistry; general pathology; and bacteriology, including parasitology and immunology.

Part II consists of examinations in surgery, including applied anatomy, surgical pathology, and surgical specialties; obstetrics and gynecology; pediatrics; neurology and psychiatry; public health, including hygiene, medical jurisprudence; osteopathic principles, therapeutics, including pharmacology and materia medica.

Part III is an oral and practical examination given under the supervision of a chief examiner who is a member of the Board and by a panel of associate examiners. Subjects covered in Part III are anatomy; physiology; pathology; osteopathic principles; therapeutics, and pharmacology; surgery; ophthalmology and otorhinolaryngology; obstetrics and gynecology; physical and clinical diagnosis; public health and communicable diseases.

Part III examinations will be given at Philadelphia, March 3-4, at Kirksville, Mo., March 10-11, and at Los Angeles, March 24-25. Applications must be filed by January 1.

These are oral examinations which the candidate may take after having satisfactorily completed the first 6 months of a 1-year internship in a hospital approved by the American Osteopathic Association for intern training. Part III is given annually at the above-named colleges.

Eligibility requirements are as follows: Part I, satisfactory completion of the first 2 years in an approved school of osteopathy; Part II, satisfactory completion of Part I and of the first two quarters or trimesters of the senior year in an approved osteopathic college; Part III, satisfactory completion of Part II and at least 6 months of a 1-year internship approved by the American Osteopathic Association. The internship requirement does not apply to candidates who took Part I prior to July, 1950.

Application must be filed with the secretary of the Board not less than 30 days prior to the examination dates. Address Paul van B. Allen, D.O., secretary, 1512 N. Delaware Street, Indianapolis 2, Indiana.

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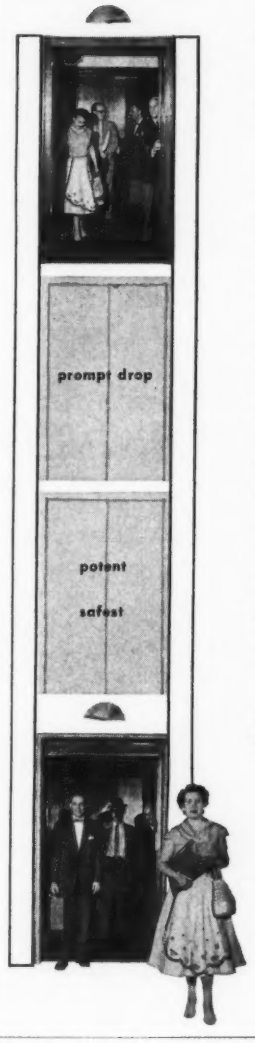

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SUMMER ACCIDENT HAZARDS*

The accident death toll in the United States is somewhat greater in the summer than at any other season of the year. Of the fatal injuries sustained in 1950-1952, 27 percent occurred during the summer (June through August), compared with 25 percent in the autumn and with 24 percent in the winter and the spring. While the seasonal peak is small, it nevertheless accounts for an appreciable excess in the actual number of deaths. In an average year, there are about 3,000 more deaths from accidents in the summer than in the spring. This heavier loss of life reflects largely the increased mortality resulting from outdoor recreation and sports, from most types of transportation, and from outdoor occupational activities.

Drownings account for a considerable part of the excess mortality from accidents in the summer. Of the more than 5,000 deaths a year from drowning in the United States, over one half occur in the three months June through August, a higher seasonal concentration than for any other cause of accidental death. Similarly, fatal injuries resulting from water transportation have their peak incidence in the summer, with two fifths of the deaths occurring in this season. Most of the deaths sustained in such accidents involve the use of small boats, and add to the toll of drownings. The other means of transportation, except motor vehicles, also cause more deaths in the summer than at other times of the year, but their seasonal peak is less pronounced.

Fatal injuries from machinery accidents increase substantially in the summer; in 1950-1952, 30 percent of these fatal injuries took place from June through August. The seasonal peak for machinery accidents reflects in large measure the increase in farm activities, with tractors and other power equipment contributing appreciably to the toll. The increased summer incidence of deaths from electric current is likewise due largely to occupational hazards. To complete the list of special summer hazards, mention should be made of lightning; although it is responsible for only about 200 deaths a year, approximately three fourths of them occur during the summer.

While motor vehicle accidents do not reach their maximum until the autumn, they far outrank every other cause of accidental death in the summer, as they do at all other times of the year. Actually, the summer toll is only slightly less than the autumn's; in 1950-1952 the proportion of deaths in the two seasons was 26 and 28 percent, respectively. The great volume of travel adds considerably to the death toll during the vacation season. It is noteworthy that the summer rise in fatal motor vehicle injuries is due entirely to nonpedestrian accidents, that is, mishaps involving collisions between automobiles, and vehicles overturning on the road, hitting a tree, or running into some other fixed object. The number of fatalities among pedestrians actually declines to a minimum in the period from June through August.

Falls rank second to motor vehicles as a cause of accidental death in the summer, not because fatal falls vary appreciably with the season, but because they have a relatively high incidence throughout the year. In 1950-1952, accidental falls in the United States took more than one and one half times as many lives as drowning (including those in water transportation) from June through August. Several types of falls, such as falls out of windows and off roofs and trees, as well as diving accidents (which are classified with falls), have a summer peak.

It is a grim fact that accidents in our country take about 25,000 lives each summer, a large proportion of which are lost because of negligence or recklessness. The toll from water sports, particularly among teen-agers, would be reduced if the ability to swim were made a part of the child's general training, and if swimmers would not attempt feats beyond their strength. Even more important than the special seasonal hazards is the motor vehicle accident problem. One of the disturbing aspects of this problem is the complacency with which the American

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, June, 1955.

people, season after season, year after year, accept the slaughter on our streets and highways. A countrywide effort is being made to arouse the nation to effective action through the "Slow Down and Live" program sponsored by the National Conference of State Safety Coordinators. This program merits our active support.

SERVICES TO CRIPPLED CHILDREN, 1935-55*

Edward Davens, M.D.
Chief, Bureau of Preventive Medicine
Maryland State Department of Health

The following review of developments in the care of handicapped children during the past 20 years has been compiled from comments of directors of State crippled children's programs in various parts of the country. A request to all State directors for a "few paragraphs" on what they would like to see emphasized in an article brought 25 replies.

These comments were impressive in revealing the quality of the directors' ideas, their obvious concern for extension and improvement of total care of handicapped children, and an indication of nationwide ferment toward better integration of services. The feeling that crippled children's programs are still in a stage of dynamic growth came through very strongly. Undoubtedly the programs are riddled with unsolved problems and glaring omissions. Nevertheless the communications from the States provoke a thrill of pride at the vigor and vision with which these are being attacked.

Many of the letters pointed out that some State crippled children's programs preceded the passage of the Social Security Act, in a few instances by many years. All, however, acknowledged that the act gave "tremendous impetus to the broader development of services for children, particularly in rural areas," through its three-way partnership arrangement involving local, State, and Federal governments.

Twenty years ago the purpose of the program was set forth in the Social Security Act, title V, part 2, section 511, as follows:

". . . enabling each State to extend and improve (especially in rural areas and in areas suffering from severe economic distress) as far as practicable under the conditions in such State, services for locating crippled children and for providing medical, surgical, corrective, and other services and care, and facilities for diagnosis, hospitalization, and after-care, for children who are crippled or who are suffering from conditions which lead to crippling . . ."

While the Federal Government has increased its share of the expenditures for crippled children's services threefold since 1940, when it spent $3,700,000 on the program, State and local governments have in the same period increased their contributions sixfold—from $5,600,000 in 1940 to $29,800,000 in 1954. However, approximately 40 percent of the State and local share in 1954 was raised and spent in only four States. Some States, with comparatively low incomes, appropriate very little more than the amount necessary to match the Federal dollars, while one or two are not putting up enough to draw the full amount of Federal aid due them.

BASIC PHILOSOPHY

Any program of medical services for crippled children needs guideposts to point direction and goals. These vary somewhat from State to State but on the whole derive from an underlying philosophy which may be stated in four basic principles:

1. The program has a humanitarian basis growing out of a normal concern for the well-being and happiness of children. This is widely recognized and accepted. Less well understood is the tremendous economic saving to the State in dollars and cents associated with trans-

*Reprinted from *Children*, July-August, 1955.

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*U. S. Patent No. 2,441,498

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in urinary tract infections

Investigators:

Flippin, H. F., and Eisenberg, G. M.: Antimicrobial Therapy in Medical Practice, Philadelphia, F. A. Davis Co., 1955, p. 40.

Findings:

Clinical studies demonstrate rapid response in cystitis and pyelonephritis, including refractory infections.

Trafton, H. M., et al.: New England J. Med. **252:** 383, 1955.

13 acute cases: 6 appeared cured, 6 markedly improved, no relapses.

36 chronic cases: 30 symptomatic improvement, often in 24 hours.

Beutner, E. H., et al.: Antibiotics Annual, 1954-1955, New York Medical Encyclopedia, Inc., 1955, p. 988.

Furadantin eradicated 29 strains (62%) of 47 isolated from 30 chronic infections.

Hasen, H. B., and Moore, T. D.: J.A.M.A. **155:** 1470, 1954.

Acute infections: 95.7% of patients benefited. Chronic infections and organic or obstructive lesions: 82% benefited.

Dosage: Average adult—four 100 mg. tablets daily, 1 tablet with each meal and 1 with food or milk on retiring.

Furadantin tablets, 50 and 100 mg., bottles of 25 and 100. Furadantin Oral Suspension (5 mg. per cc.), bottle of 4 fl.oz. (118 cc.).

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formation of potentially lifelong handicapped, dependent, unemployed persons to useful citizens. Increase in manpower resources and the consequent gain in national security is an additional consideration.

2. Primary emphasis should be directed toward application of the increasing numbers of preventive measures which medical science is providing. Programs for crippled children should never be allowed to become mere salvage operations to patch up disabilities which need never have occurred.

3. Every service needed for the total medical care and rehabilitation of the crippled child should be employed. All available private resources should be utilized and the balance financed from public funds. Continuing effort should be made to encourage individual initiative and the assumption by parents, within their ability, of responsibility for the care of the handicapped child.

4. The effectiveness of a State crippled children's program depends upon the degree to which its activities are correlated and integrated with the medical and allied professional groups, medical schools, hospitals, voluntary health agencies, and various social and educational agencies in the State and local community. A program is not and cannot be made a self-contained unit.

In applying these principles it must be remembered, as Eliot[1] has recently reminded us, "that physically handicapped children are particularly exposed to discrimination, isolation, and rejection by the community and even sometimes by their own families." Said Dr. Eliot:

"We know only too well that any child who is rejected or who feels himself deserted by his family will find the road to a healthy personality a most difficult one.

"Handicapped children then, even more perhaps than normal children, need the bulwark of family trust, affection, acceptance, and support if they are to avoid overt aggressive behavior, even delinquency, on the one hand, or the building of walls of defense by retreat within themselves on the other. This places a great burden upon all of us who have a part in guiding the future development of this program to *continue to look at our procedures and practices.* We must make sure that everything is being done that can be done to bring complete health to the children under care."

COVERAGE AND PRIORITIES

The most consistent trend nationally, as revealed in the letters from the State directors, has been the broadening of the definition of a crippling condition. During the early years of the program most States concentrated on providing services for orthopedic handicaps in conformity with the then current definition of the term "crippled." Disabilities amenable to plastic surgery such as cleft lip and palate were soon added. About 1941 several States, stimulated by Federal funds for "special projects," began to set up demonstration programs for children with rheumatic fever, rheumatic heart disease, and hearing impairment.

Then cerebral palsy, previously included under orthopedic conditions, began to receive special attention in several States, which developed projects designed to further integrate medical and educational services by providing physical, occupational, and speech therapy and medical, psychological, and social supervision simultaneously with the education of the child.

Recently this integrating trend has accelerated and has been extended to a host of new handicapping conditions. In addition to those already mentioned conditions receiving special attention in State programs today are sight conservation as well as eye disorders amenable to surgery, speech disabilities of all types with the point of reference being "communicative dysfunction," congenital heart disease, epilepsy, nephrosis and nephritis, diabetes, asthma, severe orthodontic defects, and even the clinical aspects of specific reading disability.

These trends have been accompanied by concern over what one State director described as "the emotional factors involved in not only the impact of sudden disability and the disturbance of normal growth and development of the personality by congenital or early disability, but also the physical expression of emotional disturbances which require careful and complete evaluation." As an example of the latter he cited efforts toward the de-

tection of functionally deaf or hard-of-hearing children.

Practically all of the responding State directors stressed the insufficiency of appropriations to meet the needs of at least some types of conditions. One program director pointed out that "not only has the cost of medical care per individual unit gone up tremendously within the past decade but furthermore the amount of medical care per person has also increased so that at the present time we are faced with a serious problem in meeting the financial cost for such improved care."

As a matter of fact, funds, personnel, and facilities for crippled children's services will probably always be limited in relation to actual need. This means that program directors, staffs, and advisory groups must think about spending available funds in a way to achieve the greatest benefit for the largest number of children at the least cost. In developing a balanced array of services, increasing attention is being given to priorities of emphasis stemming from sound public-health criteria rather than from sentiment. Basic criteria are:[2]

1. The number of children affected.
2. The severity or potential severity of the handicap.
3. The amenability of the handicap to medical therapy and preventive measures.
4. The likelihood of the child's becoming, if untreated, dependent, unemployable, and a permanent public responsibility.
5. The unfavorable effect of the handicap on the emotional, economic, and social status of the child and his family.
6. The extent of interference with satisfactory progress in school and future employment.
7. The cost per child to secure maximum benefit.
8. The progressiveness of the condition.
9. The availability of special personnel and facilities for treatment.

STANDARDS AND DISTRIBUTION

Undoubtedly the most significant and enduring contribution of the State crippled children's programs has been the way they have carried the torch for high standards of medical care.

For the past two decades the persistent insistence on a high quality of care has been notable. It has paid off large dividends in terms of qualifications of professional personnel; high standards of facilities used; and organization of services so that children can be moved freely from rural areas to medical centers and back, from hospitals to long-term institutions, from hospitals to their homes with all of the needed resources and services available to them at each point.

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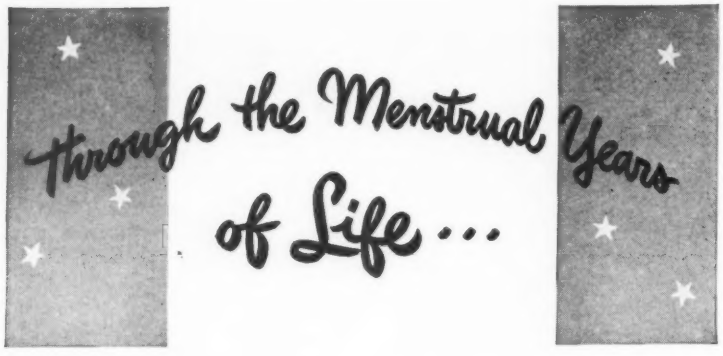
A large part of the credit for these achievements should go to the orthopedic statesmen who had the vision and drive to insist on high standards back in 1935 and before. History will also surely chalk up several points on this score for the Federal Children's Bureau. As one State director put it, "Children's Bureau consultative service to the States has promoted establishment and maintenance of standards of medical care and has indoctrinated State staffs in new and broader concepts of care of children."

In this connection another significant result has been the tendency of private agencies to adopt the standards of care set by State agencies for both treatment facilities and personnel.

Of first importance in the elevation of standards and in the development of new programs has been the use of a portion of the Federal grants for providing opportunities for special training. Federal money has been used to support courses in pediatric nursing, cleft-palate surgery, audiology, the care and treatment of epileptic or rheumatic-fever patients, and various aspects of physical therapy, as well as to provide for medical social work field practice in agencies for crippled children.

One of the outstanding characteristics of medical practice in the past 50 years has been the trend towards specialization and urbanization with a consequent maldistribution of personnel and facilities,



THE frequency with which the menstrual life of so many women is marred by functional aberrations that pass the borderline of physiologic limits, emphasizes the importance of an effective uterine tonic and regulator in the practicing physician's armamentarium.

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especially for complex diagnostic problems and long-term care. Medical specialists, diagnostic facilities, laboratories, and the other impedimenta of modern medicine have congregated in large metropolitan centers resulting in a geographic roadblock for the crippled child who lives in a rural section.

"One very certain value of crippled children's services has been their ability to reach children in rural areas who otherwise would be neglected," was but one of a number of such comments from the program directors. Nevertheless, the inherent difficulties imposed by long distances in providing complete and continuous care continue to be formidable. Said one comment from the West:

"Many of our children are 400 miles away from the closest center. If your children in Baltimore had to travel halfway to Chicago for treatment, you would get some idea of what we are up against. The problem of followup from the center is difficult and we are finding that most of our followup must be done through public-health nurses who have received in-service training in this field. The problem of distance in the Western States has never been satisfactorily solved. The travel costs for the State board of health personnel are very large and the time consumed cuts down on the efficiency. Since we have no medical school in the State, we use several out-of-State for postgraduate training."

Some States are attacking this problem through the establishment of regional and district offices.

INTERPROFESSIONAL APPROACH

Nearly all of the communications from the States produced emphatic evidence that the team approach is perhaps the dominant trend at the moment in organization of services.

State after State described plans for bringing together a group of specialists to work as a team in a crippled children's clinic or center. More and more we find a pattern in which a medical specialist, such as an orthopedist, and the pediatrician, medical social worker, public-health nurse, and physical therapist work together to evaluate the problem and formulate a plan of continuous care. In many instances a special educator, either in person or through appropriate communication, becomes a member of the team. For older children the vocational counselor from the State Vocational Rehabilitation Service joins in the task of making a long-range plan.

The postclinic conference is rapidly becoming a popular and effective way of reaching a deeper understanding of all the problems imposed by the disability and helping parents to plan to use all community resources in their solution.

As community mental-health organizations and child-guidance clinics increase in number, a close relationship is being established with them by crippled children's centers and clinics. Thus they can bring the skills of a team of psychiatrist, clinical psychologist, and psychiatric social worker to bear on the problems of those crippled children in whom difficulty in accepting needed treatment or excessive emotional disturbance compounds the underlying problem of physical handicap.

In the case of the subtle and complex problems associated with hearing impairment and speech disorders, the ball is now usually being carried by a skilled and smoothly functioning trio of otolaryngologist, clinical audiologist, and speech therapist, who work closely with the pediatrician, the public health nurse, and the medical social worker.

The field of sight conservation still seems to be lagging behind this trend. Better teamwork needs to be worked out between opthalmologist and optometrist before the great bulk of the difficulties can be overcome, especially in rural areas.

Many of the directors' letters expressed great enthusiasm for the new approach to total care of infants with cleft lip and palate. Nearly half the program directors referred to the development of an integrated cleft-palate clinic. One rather detailed comment illustrates how far interprofessional collaboration has advanced:

"The speech pathologist begins counseling the parents of the cleft lip and/or palate child at the time of the first application to the Crippled Children's Division. He sees the parents on the same day that the child is examined by the

plastic surgeon, which is usually the day before hospitalization for the primary closure, with the emphasis usually being on the problem of the child's future speech. Contact is then made by the Division's medical social consultant and the on-going care begins.

"At the age of about 4 years, the child is sent to the university dental school for dental X-rays, head plates, and wrist films, at which time molds of the mouth are also made. At this time the child is also seen by a pedodontist. By then the speech pathologist has made his evaluation, the medical social consultant has followed the case closely, and the plastic surgeon has seen the child yearly to suggest any operative procedures that may improve the cosmetic effect.

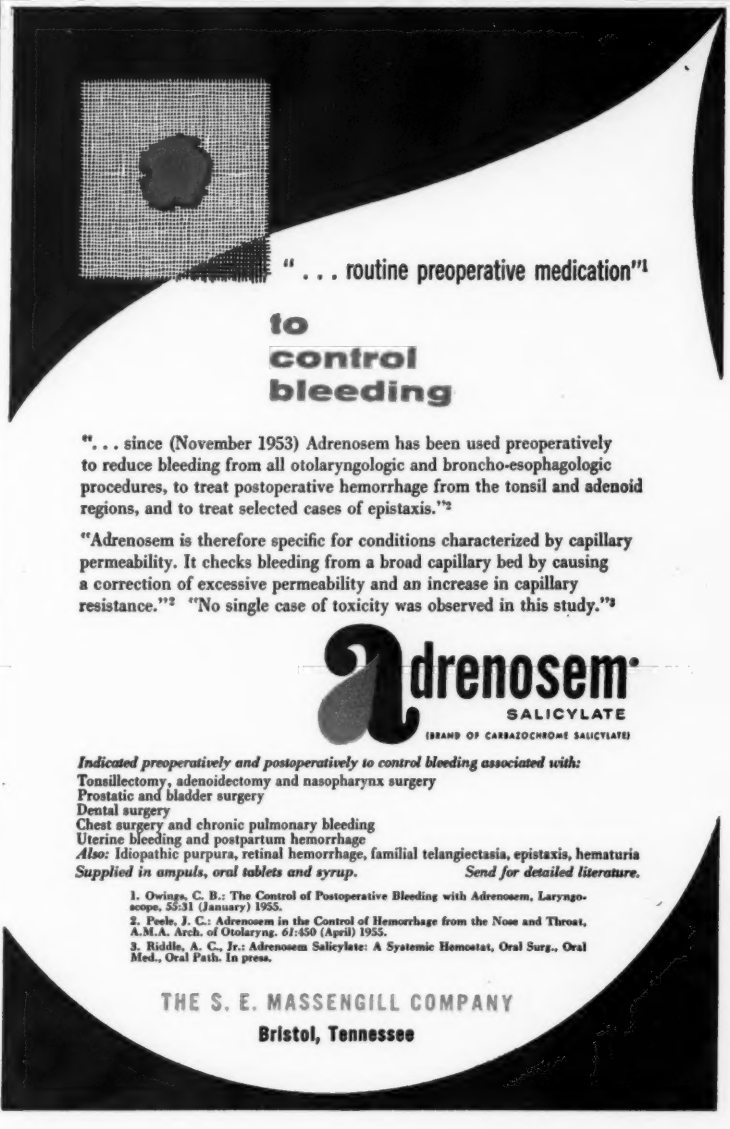
"After the age of 5 years the child, accompanied by his parents, is examined by the plastic surgeon, speech pathologist, representatives of the dental school who had performed the original examination, plus an orthodontist and prosthodontist from private practice. A pediatric examination is given and after each specialist has had an opportunity to study the patient's history, examine the patient, and review the dental films and molds, a case conference is held at which time the orthodontist, prosthodontist, plastic surgeon, pediatrician, nursing consultant, and social worker discuss with the speech pathologist any method at the disposal of the respective specialist which may improve the child's speech. There is a free flow of ideas among all of the specialists to determine what surgery, orthodontia, or prosthodontia may be necessary to give a good cosmetic effect and normal alinement of the teeth, as well as any type of device which will improve the child's speech.

"For the next 15 years the parents are instructed from time to time by the medical social worker on how best to coordinate all the services. The nursing consultant sends the composite data to the public-health nurse in the child's own community. She then coordinates the followup care and urges the family to get all necessary work done so that the orthodontist and prosthodontist may properly carry out their function."

ACCENT ON PREVENTION

The people who wrote Title V of the Social Security Act back in 1935 had the vision to see the great importance of applying the principle that an ounce of prevention is worth a pound of cure. This idea is incorporated in this section of the act by the wording "or who are suffering from conditions which lead to crippling."

In the early years of the program relatively little attention was paid to preventive possibilities possibly because of the preoccupation with taking care of the backlog of severely handicapped children. Even in 1955 much remains to be done in putting into day-by-day practice the close relationship which should exist between maternity, infant, and preschool, and school health programs on the one hand and crippled children's programs on the other.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In recent years, much more attention has been focused on the preventive aspects of crippling, partly because of stimulation from the Children's Bureau through a variety of "special projects." The concept of different levels of prevention is now widely accepted. "Primary" prevention for example would be pasteurization of milk to reduce the possibility of bone tuberculosis; smallpox, diphtheria, and other active immunizations to prevent various disabilities; the provision of cod-liver oil to avoid rickets; and a host of other procedures. "Secondary" prevention refers to the planned and systematic application of early case-finding techniques, prompt and definitive diagnosis, and arrangement for treatment and meticulous followup so that the "condition which leads to crippling" is taken care of before handicap results or becomes severe. Early diagnosis and care of rheumatic heart disease and hearing impairment are typical examples of this approach. Such preventive efforts require careful organization of services, constant effort to follow through each case, and close and cordial collaboration between the crippled children's program and the maternal and child-health and school health programs, voluntary health services, medical schools and teaching hospitals, and other community services.

Perhaps the greatest challenge facing crippled children's programs at this time is subtle, sustained, and complex cooperative community action needed to realize fully the goal of "secondary prevention."

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NEW TREATMENT METHODS

State crippled children's programs have been responsive to the many new discoveries in medical diagnosis and treatment which have become available.

Rickets as a cause of crippling was already on the way out in 1935. Improved case-finding procedures and additional treatment facilities as well as pasteurization of milk and new antibiotics have dramatically reduced bone tuberculosis. Chemotherapy and antibiotics have also greatly reduced both the number and the severity of cases of osteomyelitis, an inflammation of the bone marrow. The new Salk vaccine promises to control poliomyelitis.

In the field of childhood cardiac disability the advent of sulfadiazine and penicillin have made possible the control of rheumatic heart disease, while the development of remarkable new operative techniques have opened the way to habilitation of over half of the cases of congenital heart disease.

During World War II many remarkable new ideas and procedures were developed which have boosted the whole field of rehabilitation. One of the most remarkable examples is the creation of a new medical specialty, clinical audiology, which has practically revolutionized previous thinking about "deaf" children and made it possible to provide auxiliary methods of communication to nearly all youngsters with impaired hearing for whom treatment is started early enough and maintained with the necessary intensity.

One of the most exciting recent developments has been the improved approach to upper- and lower-limb prosthesis, resulting from research spearheaded by the National Research Council and characteristically based on the team approach of orthopedist, physical therapist, and prosthetist.

Other exciting examples could be added to this list. But there is also another side of the coin, for sometimes the wonder drugs and improved methods of obstetrics and care of premature infants result in the survival of severely disabled infants. Nearly all program directors expressed concern about the increasing numbers of children in the categories requiring custodial care or institutional type of habilitation service. The solution to this problem would seem to be a "must" for the next decade.

THE IDEAL PROGRAM

Of necessity this cursory survey has simply highlighted certain significant advances. Crippled children's services still have far to go to reach the goal intended in the basic enabling legislation. Currently the Child Health Committee of the American Public Health Association is developing a number of "Statements on Recommended Practices in Programs for Crippled Children." When published these will be helpful in crystallizing current thinking on the best approach to the several types of handicaps.

Meanwhile Kobes[3] has recently offered us a definition of what a program should be:

"An ideal crippled children's program is one which has the broadest possible range of high-quality medical care and related services available to meet the needs of children who have a broad spectrum of medical diagnoses. These medical categories should perforce be those which are characterized by long duration of the effects of the handicapping condition and thereby cause an undue amount of drain on the family's energies and economic resources. Adequate payments should be made to all who render services, whether they be full-time staff members or part-time consultants giving clinical services. All families should make payments toward the cost of care within their ability to pay. All personnel should meet the highest possible standards of professional ability and performance. The health and medical-care services needed to obtain the best clinical results for each child should be made available, and the social, emotional, educational, and vocational-training aspects of each child's needs should also be the concern of those who have the responsibility of administering the State's crippled children's program. Whenever it is necessary to do so to insure a wide range of capable services, the State should provide training programs to help fill any gaps which may be present. Existing resources should be used to the fullest extent and without duplication of services. They should be improved whenever necessary to meet the most desirable standards of care, and only when existing resources are defi-

nitely proved to be lacking should completely new facilities be constructed. There needs to be sharing of resources among communities, even to the extent of working out interstate arrangements when it seems undesirable to make available for one State a service which may be used but rarely. Only when we know that we have a program which will bring the maximum number of physically handicapped children into our society as useful, productive members can we feel that our job is nearing its highest level of success and accomplishment."

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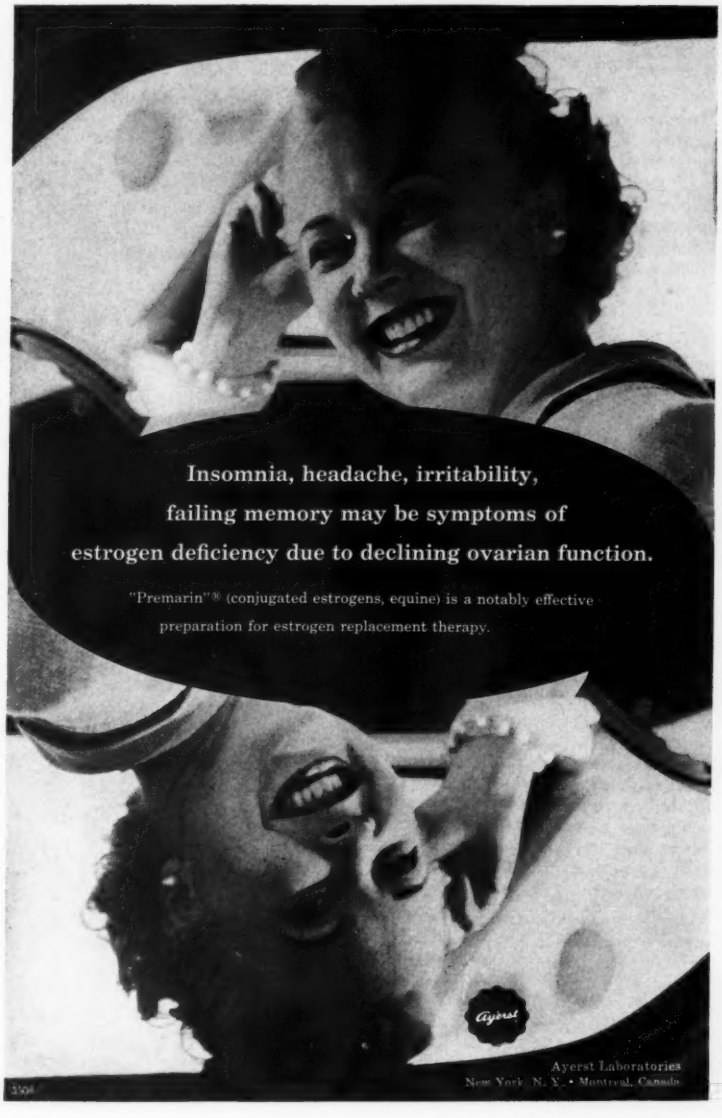
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MEDICAL EDUCATION IN TRANSITION*

Julius B. Richmond, M.D.,
Professor and Chairman, Department of Pediatrics, State University of New York, College of Medicine, Syracuse

Medical education is in transition. Dr. George Berry, dean of the Harvard Medical School, has described this transition succinctly as a shift from an emphasis on the "patient as a disease" to the "patient as a human being."[1] Dr. Alan Gregg, vice president of the Rockefeller Foundation, quoting an old French aphorism, has called this a rediscovery of the fact that "there are no diseases; there are only sick people."[2]

That considerable dissatisfaction existed with medical education on the part of medical educators and the consumers of medical care by the end of World War II has been abundantly demonstrated by the subsequent introduction of many changes in the educational process. Some of these changes will be outlined further on in this article. They came about in response to a need for new perspectives in medical education which may be understood best, perhaps, in the light of the historical development of the modern medical school.

As we know it today the medical school is a relatively new institution. At the turn of the century the major part of medical education was provided by numerous proprietary schools scattered throughout the country. Although the trend toward a 4-year curriculum was already underway, the publication of the Flexner report[3] in 1910 accelerated the movement. Consequently, many schools were brought into the fold of universities, a standardizing agency—the Council on Medical Education of the American Medical Association—came into being, and the proprietary schools gradually disappeared. The licensing boards of the various States also sped up this trend.

Along with the building up of the 4-year curriculum rapid advances in the biological and medical sciences were taking place. Accepting their full responsibilities to society and to their university identification, medical schools not only carried out their teaching functions, but also turned themselves into strong research centers. The developments which emerged are now common knowledge. The prolongation of life, the reductions in maternal and infant mortality, the decline of child mortality from infectious and nutritional disorders are but part of the rich harvest of this era.

Because of the relatively uneven development of medical schools, the first half of the 20th century was characterized by an emphasis on raising standards and incorporating new scientific knowledge within the 4-year curriculum. The rapid advances in knowledge presented a real problem, for more content kept being added to the curriculum with each passing year while other subject matter rarely was deleted. Difficult though this was for faculties, it was even more

*Reprinted from *Children*, September-October, 1955.

rigorous for the student. Upon one point there seemed to be general agreement: medical students had attained a degree of technical competence never before achieved.

THE CRITICISMS

The major disruptions in medical education and practice associated with World War II and the increasing expectations from medical practitioners on the part of the community stimulated a reevaluation of medical education. This brought forth the following general criticisms:

1. Teaching is too compartmentalized. The student is taught by groups of specialists, preclinical or clinical. He has too little opportunity to observe the patient as a whole or to observe faculty members who present an approach to the total patient.

2. The student is not brought into contact with patients during the first 2 years of the medical school curriculum. The strong motivation with which many students enter upon the study of medicine is therefore unexploited during the first 2 years.

3. Content about the impact of social and psychologic forces on the individual has generally not been adequate to prepare the student fully for a constructive role in the community on entering practice. When such teaching has been attempted, it has tended to be too didactic to be meaningful to the student; the clinical setting has been used inadequately. An opportunity for the student to develop a background for his own social thinking and insight into his own personality development has been lacking generally.

4. Insufficient emphasis has been put on preventive medicine and the maintenance of health. The doctor's role in developing a positive health program for our population should complement his concern with the study of disease.

MEETING THE PROBLEMS

During the past several years, medical schools individually and collectively have been attempting to deal with these problems. The American Medical Association and the Association of American Medical Colleges have jointly sponsored an elaborate survey of medical education, which has now been completed.[4,5] The American Academy of Pediatrics has sponsored a study of pediatric education followed by annual conferences on educational problems.[6] The Association of American Medical Colleges and the American Psychiatric Association have jointly sponsored a conference on psychiatry and medical education.[7] The Association of American Medical Colleges has supported a conference of professors of preventive medicine.[8] Annual teaching institutes concerned with curriculum problems and teaching methods among the various departments of medical schools are currently under way. Conferences on premedical education and on administrative medicine have also been held. The congresses on medical education and licensure sponsored by the American Medical Association and the annual meetings of the American Association of Medical Colleges have provided opportunities for the exchange of ideas on educational planning.

Individual medical-school faculties have been vigorous in reorganizing and reintegrating their curricula. Thus while the first half of the 20th century brought about a traditional and relatively static curriculum, the second half promises to be one of increasing flexibility and experimentation. However, the experimentation which is now under way is generally characterized by a healthy maturity and skepticism. In general a real effort has been made to build into these programs opportunities for evaluation of their effectiveness, difficult though any evaluation of an educational process may be.

Lest their be any concern about possible loss in the teaching of scientific medicine it is important to emphasize that the gains which have been made in this area are being carefully protected. There is no desire to have the physician less well equipped scientifically than he has been in recent years. However, some of the new flexibility can be attributed in part to the increasing realization that undergraduate medical education is no longer the terminal education of the physician. Graduates generally proceed with at least 1 year of internship while increasing numbers pursue specialty

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training for an additional 3 or more years. Thus the undergraduate medical curriculum is only a segment of the training of physicians today. It is folly, therefore, to add information increasingly to the curriculum without reevaluating content with the objective of providing the student with a body of knowledge which his cerebral cortex can realistically deal with and absorb.

NEW PROGRAMS

In order to attempt to prepare the modern physician for his social responsibilities to the community and to deepen his understanding of the patient as a person, a variety of programs are in progress—more than can be described in the space of this article. In one instance these efforts have involved a thoroughgoing reorganization of the curriculum of the medical school. In other instances the attempt has been to introduce new courses within the conventional 4-year curriculum. In general these programs may be summarized as follows:

1. A complete reorganization of the medical-school curriculum, as exemplified by the program at the School of Medicine of Western Reserve University. In this program curriculum reorganization has resulted in integrated teaching of the conventional subject matter. The faculty has bridged the traditional departmental compartments to present to the student a view of human biology whole. Thus structural, biochemical, and physiological relationships are taught concomitantly according to an interdepartmental plan by faculty members without regard to their departmental affiliations. As a consequence of this curricular reorganization, considerable revision of the physical plant of the medical school has been necessary, since the student studies in a laboratory of his own rather than in a sequence of departmental laboratories.

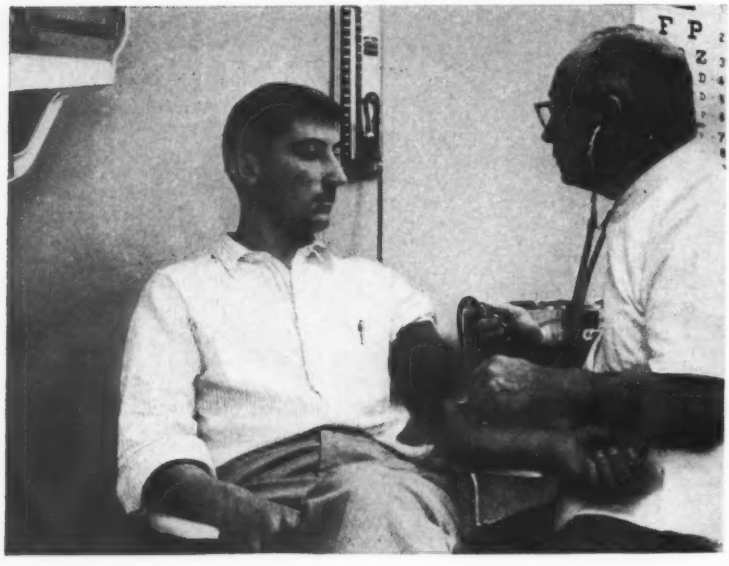
The student early in his freshman year begins to work with a family, focusing on the care of a baby. His contact with the family starts in the prenatal period and carries through and after delivery. Guided by a preceptor, his relationship with the family continues over the 4 years of medical school. He learns the role of social workers and other personnel with whom the physician works in planning for the care of patients in the community. Thus an opportunity is provided at the outset for him to develop relationships with patients and to utilize the motivation which presumably brought him to the medical school.

In such a program a strong commitment on the part of the faculty to such a complete reorganization of curriculum and teaching methods is essential for success. Other medical schools have not yet undertaken such complete revision, but the program is being observed with considerable interest and enthusiasm by medical educators today.

2. Home care programs. Several medical schools in the United States have for a number of years had programs centering around the care of patients in their own homes. Through them students are provided an opportunity to see patients in their social and cultural settings and community relationships and to develop a greater appreciation of the problems associated with their medical care.

The effectiveness of such programs is generally related to the time available and the skill and interest of the teaching faculty. Unfortunately because of the fact that students are often assigned to these programs for short periods of time, usually in the junior and senior years, they have little opportunity to observe continuity of care and to develop the deeper insights into patients' problems which come from repeated contact.

3. Outpatient programs designed to provide continuing experiences with patients. The Cornell and Colorado Medical Schools have attempted to assign fourth-year students to extended periods in the outpatient departments and to provide them with a general assortment of patients in order that they may have continuing experiences in providing comprehensive care. This contrasts with the traditional system of rotation through the usual specialty clinics of the medical school. The student has an opportunity to experience the same medical problems which he would ordinarily see in specialty clinics, but at the same time to develop a continuing relationship through which he can deepen his insights into the social, cultural, and personality patterns of the individual and the family. This type of



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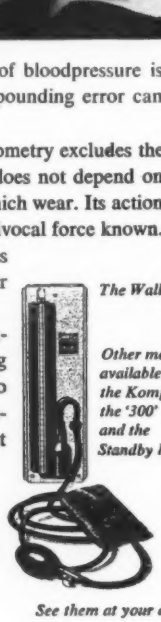

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program involves considerable reorganization of outpatient facilities and reorientation of staff. In general, additional staff is also desirable.

Because of the increasing interest in prepayment medical plans, group practice, and labor health plans, such settings are also being utilized for the teaching of comprehensive medical care. They make it possible to place a heavy emphasis on preventive health services, which conventional outpatient facilities often do not provide.

4. Family-study programs. Some medical schools have developed programs providing for long-term study of a sampling of families. Although some of these programs have been established primarily for research into the medical, social, and psychological aspects of family life, students are increasingly being integrated into them. Many programs of this type provide an opportunity for exposure to disciplines as varied as microbiology, immunology, epidemiology, cultural anthropology, social work, psychology, sociology, and the various medical specialties. Some of them focus on providing medical care for a specific member of the family. The care of well babies has been a popular way to give students an opportunity to view family life and its impact on infant and child care. Departments of preventive medicine and pediatrics have been particularly active in supervising such teaching.

5. Programs centering around an understanding of the medical-social problems of the patient. In some medical schools the student is introduced during his first year to the problems which families experience in obtaining and utilizing medical care. Some of these programs, as at the University of Pennsylvania Medical School, have continuity through the 4 years of the medical school; in other instances they are limited to a single year. In these programs the student obtains information from families concerning their medical problems, occupational health problems, and their feelings about the medical care which they are receiving. The students' observations about the family and its degree of integration within the community are discussed in seminars with physicians and social workers. In some schools sociologists, cultural anthropologists, and psychologists have also been drawn into these programs.

6. Earlier introduction of the teaching of psychiatry. In the last decade medical schools generally have introduced psychiatry into all 4 years of their curricula. Although in many instances such teaching in the first 2 years is didactic, it has nevertheless provided students with some knowledge of the behavioral sciences, thus better preparing them to deal with patients clinically. More effective, perhaps, is the utilization of a clinical setting for such teaching, a practice which is gradually becoming more common. In whatever setting, courses in psychiatry also help the student develop deeper insight into his own feelings and attitudes.[8]

7. Courses in human development and human biology. Some schools have attempted to provide students with a better understanding of the total development of man through a dynamic presentation of human growth and development in physical, intellectual, psychologic, and social terms. These courses may be didactic presentations which are largely interdisciplinary and interdepartmental in nature, or they may center around a longitudinal growth study program such as that of the Child Research Council, University of Colorado School of Medicine, in Denver, or in some instances around the observation of the growth and development of an individual baby and family, as in the previously mentioned family-study programs.

8. The introduction of psychology, sociology, and cultural anthropology into the curriculum. Many medical educators have felt for some time that students will learn more about the social, psychological, and cultural background of people if persons with highly specialized backgrounds in these fields are added to the faculties of medical schools. Therefore we are increasingly observing the addition of sociologists, psychologists, and cultural anthropologists to medical-school faculties. Their roles vary considerably from school to school, ranging from participation in research projects to the conducting of individual courses. Perhaps

the major significance of this development has been the availability of these uniquely oriented faculty members to the remainder of the faculty.

An interesting sidelight to the development of these programs is the interest in evaluating their effectiveness. Although in years past attempts at evaluation of educational processes were regarded as rather futile, research advances in the fields of sociology and psychology render such undertakings more feasible today. Current experimentation in evaluations seems to have a favorable effect on the educational processes and, hopefully, may provide objective measurement of whether programs are really accomplishing what they have been set up to achieve. Evaluation processes, however, are laborious and expensive and are still highly experimental.

SOME WORDS OF CAUTION

Although the latter half of this century seems destined to be marked by experimentation in medical education, some words of caution are in order. It would be unfortunate if the idea became prevalent that change in educational process can be accomplished by a mechanical reorganization of curriculum or by transplanting courses which have proven effective at other schools. In the last analysis, what and how a student is taught reflects the orientation of his entire faculty. The effective teaching of the social and psychological aspects of patient care is a goal for which the entire faculty must assume some responsibility. Fortunately, a core of medical educators exists who are excited about the possibility of giving students a broader orientation.

No matter how well integrated the faculty, however, the integration of medical knowledge and of social and psychological understanding of patients must ultimately be accomplished by the student individually. Therefore, in addition to a broadened curriculum, the personality and intellectual ability of the student are important factors. This means that problems of student selection emerge as primary in this whole process.

Unfortunately little is known concerning medical-student selection although each school is attempting to study the problem in its own way. Considerable concern exists about the effects of the increasing cost of medical education in discriminating against prospective students from the lower-income portions of our population.[8]

Another note of caution is in order. As has been indicated herein, during the course of his medical-school career the student is increasingly being shown by example the effectiveness of comprehensive care for patients. Consequently, the medical-school program tends to present a somewhat idealized demonstration of community resources and available personnel. This picture is often not entirely in accord with the realities of professional practice for in most communities available facilities are not comparable to the medical-school setting. To obviate feelings of disillusion and despair when the student enters practice, it becomes important that the faculty help him to learn to deal with limited goals. At the same time he should be prepared to aid in the community's planning to provide more adequate services.

Medical-school programs such as have been described here are becoming more and more costly. No longer can the schools depend solely upon individual practitioners in the community to carry on the instruction involved. As a consequence they have been steadily enlarging the size of their full-time faculties. If the community desires to have more effective physicians, it will of necessity have to find a means of supporting these enlarged programs. A prerequisite for obtaining the needed faculty members is support for potential faculty members during their extended periods of training. This is increasingly being provided through grants from public agencies, from private foundations, and from fund-raising efforts for medical schools.

Out of the newer developments in medical education more effective health services to our Nation should become available without sacrifice of the rich tradition of our medical schools. In the words of Dean Berry, "Scientific medicine must become comprehensive medicine, yet not become thereby any less scientific."[1]

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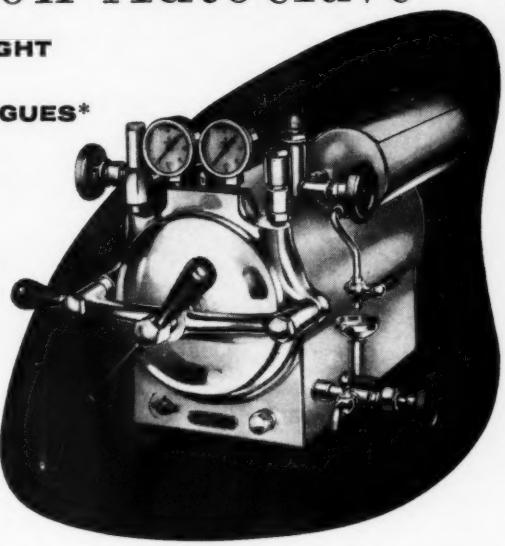

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CARDIOVASCULAR SURGERY. Studies in Physiology, Diagnosis and Techniques. Proceedings of the Symposium held at Henry Ford Hospital, Detroit, Michigan; March, 1955. Edited by Conrad R. Lam, M.D., Surgeon-in-Charge, Division of Thoracic Surgery, Henry Ford Hospital. Cloth. Pp. 543, with illustrations. Price $12.75. W. B. Saunders Company, West Washington Square, Philadelphia 5, 1955.

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CLINICAL ROENTGENOLOGY. Volume III. The Lungs and the Cardiovascular System Emphasizing Differential Considerations. By Alfred A. de Lorimier, M.D., Radiologist, Saint Francis Memorial Hospital, San Francisco, California; Consultant in Radiology for the United States Army, at the Letterman Army Hospital; Consultant in Radiation Therapy for the United States Public Health Service at the U.S. Marine Hospital, San Francisco, California; Formerly, Commandant, The Army School of Roentgenology; Henry G. Moehring, M.D., Radiologist, Duluth Clinic, Duluth, Minnesota; Formerly, Director, The Army School of Roentgenology; and John R. Hannan, M.D., Radiologist, Cleveland, Ohio; Radiologist, Lake County Memorial Hospital, Painesville, Ohio; Formerly, Director, Medical Training, The Army School of Roentgenology; Associate Professor, Diagnostic Roentgenology, The Frank E. Bunts Educational Institute, Cleveland Clinic Foundation; Staff, Department of Roentgenology, Cleveland Clinic Foundation. Cloth. Pp. 508, with illustrations. Price $20.50. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1955.

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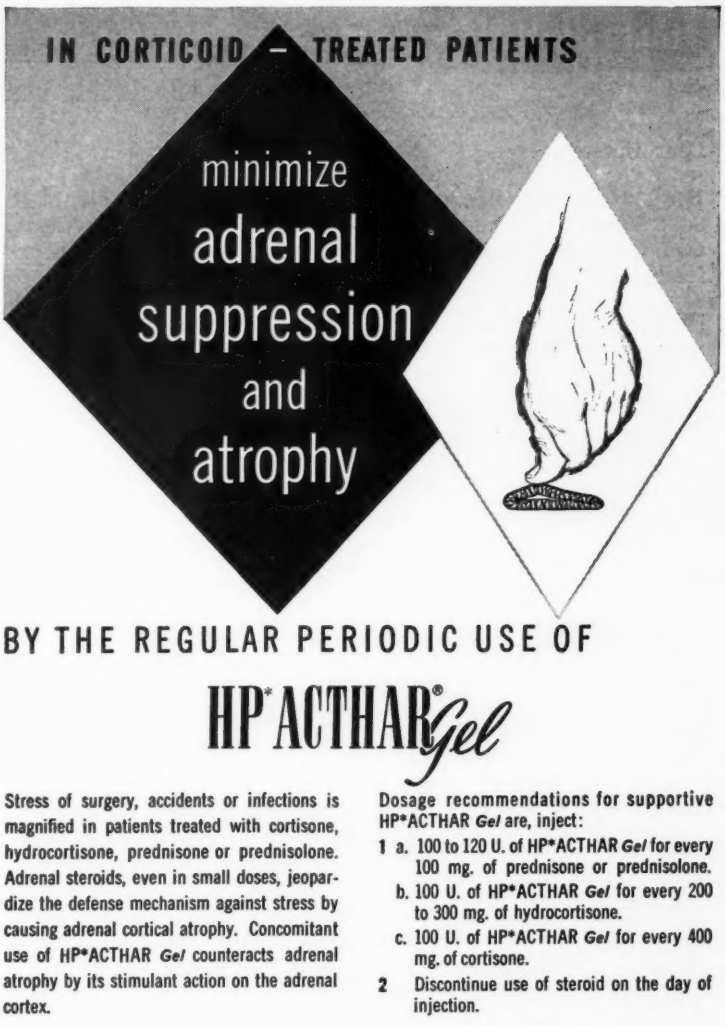
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1. Jeans, P. C., and Marriott, W. M.: Infant Nutrition. St. Louis, C. V. Mosby Co., 1947, Ed. 4, p. 364.

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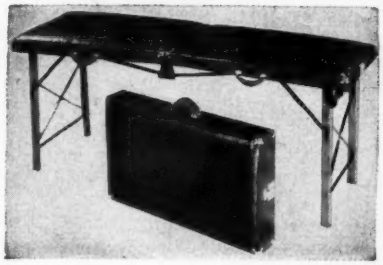

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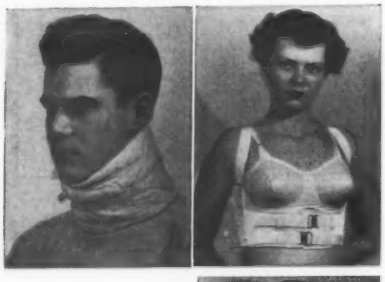

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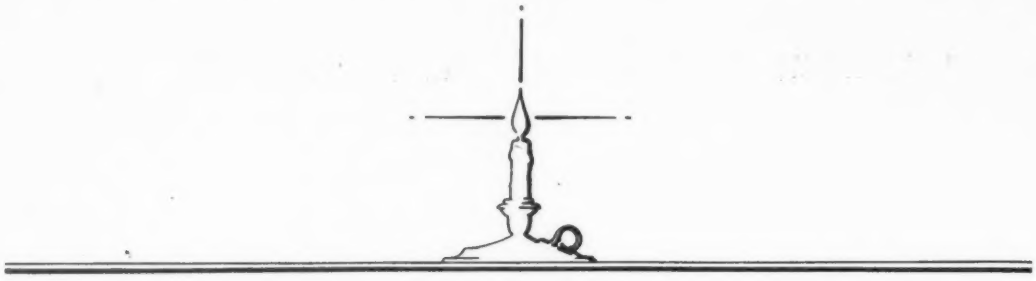

DIOGENES was a Greek, and a philosopher. He belonged to the school of cynics. Which means that he associated with a group of men who didn't believe in practically anything.

Now among the many things Diogenes didn't believe in was an honest man. But he wanted to give every one a chance, so he roamed the highways and byways of Athens with a lantern which he would flash on the faces of the passers-by, endeavoring to find in their features some sign of integrity.

Nothing ever came of his labors. (According to him). But because he was a philosopher and a reasonably fair man, Diogenes admitted that it *could* be his lantern. And so, he complained, "If only I could have a better light . . ."

And many people tried to give Diogenes a better light . . . If you're as old as sixty, you may remember that a fellow named Welsbach thought up a contraption that you fitted over a gas-jet . . . Then there was that young fellow, Tom Edison, who worked out the electric light bulb. And another genius planned a new type of light called fluorescent . . . But none of them succeeded in inventing a light that helped Diogenes.

Then a man comes up with still another kind of light—a poor sort of thing. It was a little blob of wax, wrapped around a piece of string. And one Christmas Eve he took it over to Diogenes' home. There the old fellow was, visiting with his cynic cronies, and saying as how "No, definitely not! There just wasn't such a thing as an honest man." And the cynics all nodded their heads in agreement. Then the man came in with his candle, and he lighted it. And Diogenes could hardly believe his eyes! Because as the soft, mellow beams shone out from the Christmas candle and fell on the faces around him, Diogenes could see, just as plain as could be, that there wasn't a really dishonest man in the entire gathering.

◆ ◆ ◆

And ever since then, at Christmas time, the little Christmas candles have made fools out of cynical people by proving that it is seldom that you can find a human being who, 'way down deep inside, isn't essentially honest . . . if you'll only look at him in the proper light.

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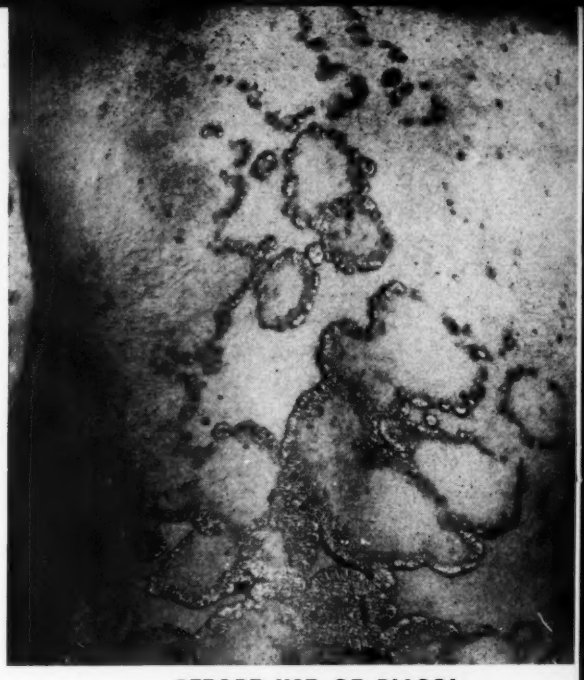

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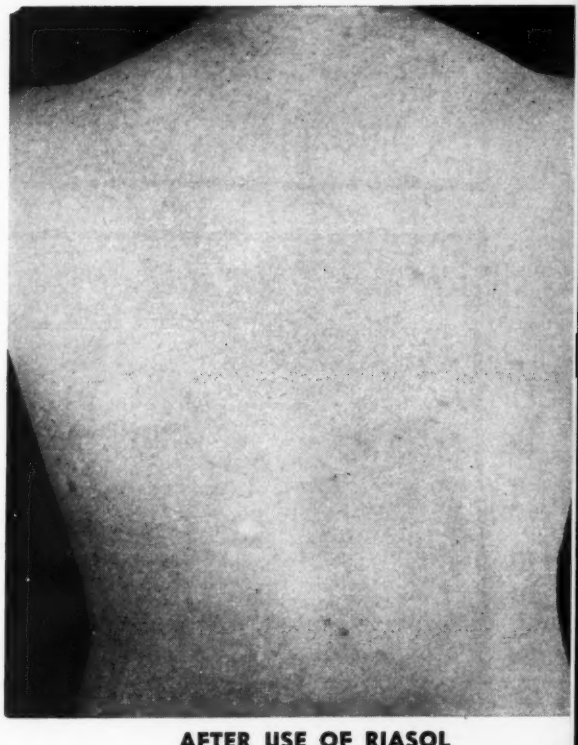

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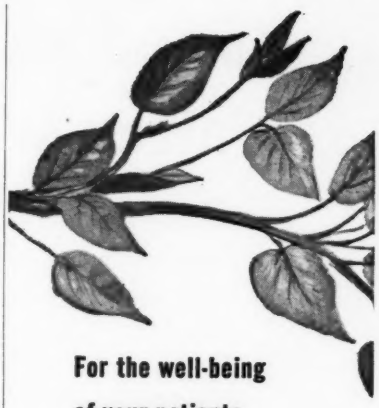

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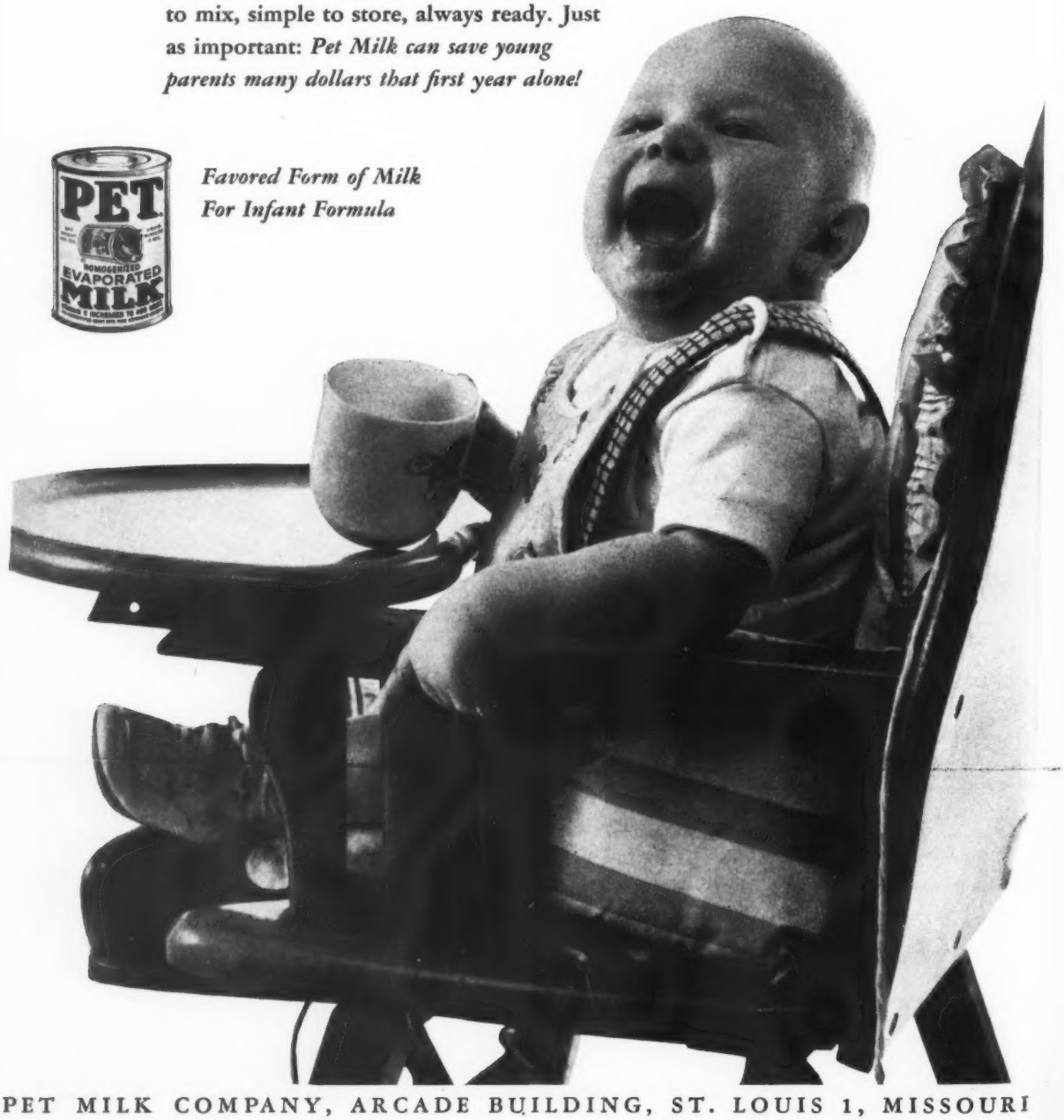

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